VOL. XXVIII. NO. 2 DECEMBER, 1934

PROCEEDINGS *of the* ROYAL SOCIETY OF MEDICINE

LONGMANS, GREEN & Co. LTD
39, PATERNOSTER ROW, LONDON
NEW YORK · BOMBAY · CALCUTTA · MADRAS

A combination of vitamins A, B and D, together with Iron and Manganese in a palatable malt vehicle.

Supplied in cylindrical glass jars containing approximately 1-lb.

AN effective means of supplementing the vitamin content of the diet is provided by this standardized combination. One teaspoonful of Irradex contains at least as much vitamin A as good quality cod-liver oil, and five times as much vitamin D. Vitamin B is provided by a standardized extract of wheat germ. Irradex is specially indicated for children or adults who are underweight, have little appetite, or who have not regained healthy vitality after some acute infection.

The moderate secondary anæmia which so frequently accompanies under-nourishment or general debility is another indication for Irradex.

During pregnancy and lactation, or other period of stress, Irradex is especially indicated. It may well be prescribed for its nutritive effect following operative procedure.

FORMULA

Each fluid ounce contains:

Liver Oil (100 A) — — —	5 mins.
Viosterol (250 D) — — —	10 „
Vitamin B Extract — — —	25 grs.
Iron and Ammonium Citrate —	4 „
Manganese Citrate (Soluble) —	¼ gr.

In a palatable base containing Malt Extract.

IRRADEX

Parke, Davis & Company

50, Beak Street, London, W.1

Laboratories: Hounslow, Middlesex. *Inc. U.S.A., Liability Ltd.*

Clinical Section

President—E. G. SLESINGER, O.B.E., M.S.

[*October* 12, 1934]

Collapse Bronchiectasis in a Child.—R. W. B. ELLIS, M.D. (for E. A. COCKAYNE, M.D.).

A. W., female, aged 5 years and 8 months. Normal infancy; parents and one younger brother well. No previous illnesses except chickenpox at age of 2, and a "cold and cough" at age of 18 months, followed by slight winter cough each year subsequently. The child is entirely free from cough at present. She has had good general health, and was referred to hospital after routine examination by the school doctor.

On examination.—A healthy-looking child with good colour, weighing 35½ lb. Height 40 in. No cyanosis or dyspnœa; ? very early clubbing of fingers. There is no fœtor of the breath. The trachea shows considerable displacement to the right, and the dorsal spine a slight scoliosis with the concavity to the right. Diminished movement of right chest.

The apex beat is palpable in the third and fourth intercostal spaces half an inch to the left of the sternum, and the cardiac impulse is visible in the second, third and fourth spaces 1½ in. to the right of the mid-line. The area of cardiac dullness corresponds with the cardiac impulse. There is no impairment of resonance over the lungs except over a small area at the right base. The liver dullness reaches to the fifth space on the right. Air entry greatly diminished over whole of right lung; breath sounds harsh in the right axilla, and tubular below the angle of the right scapula. Occasional râles over whole of right lung posteriorly. Tonsils moderately enlarged. Abdomen: nothing abnormal discovered.

Mantoux test (1 in 1,000) strongly positive.

Stomach wash-out examination negative for tubercle bacilli; no evidence of tuberculosis at post-mortem of injected guinea-pig.

Skiagram of chest (1.7.34) showed displacement of heart to right, with a triangular shadow across the right cardiophrenic angle (fig. 1).

Lipiodol injection (4.7.34) showed saccular dilatation within the triangular shadow. From the division of the main bronchus, this shadow is seen to represent the whole of the collapsed right lower lobe (fig. 2).

Treatment.—The patient was treated with repeated inhalations of carbon dioxide, and though these caused considerable hyperpnœa it was not possible to re-expand the collapsed lobe.

COMMENT.—This case is shown to illustrate the existence of saccular bronchiectasis, involving the whole of a collapsed lower lobe, with almost entire absence of cough, sputum, or any detectable malodour of the breath. There is no interference whatsoever with the general health.

The question arises as to whether these cases are to be regarded as congenital in origin, or as examples of bronchiectasis arising in a lobe that has collapsed after

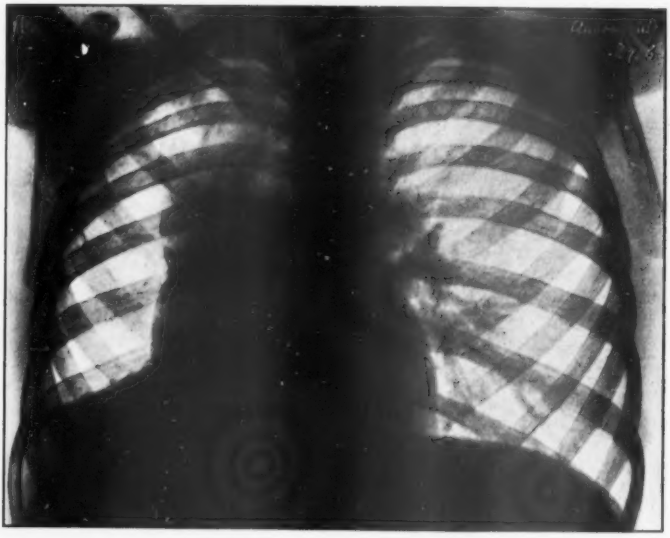


FIG. 1.—Triangular shadow across right cardiophrenic angle; heart displaced to right; elevation of right crus of diaphragm.

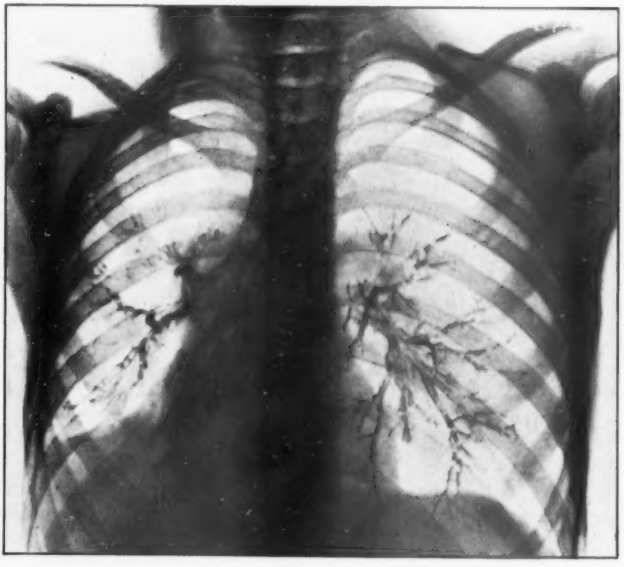


FIG. 2.—Lipiodol injection: Saccular bronchiectasis within collapsed right lower lobe; trachea displaced to right; division of main bronchus shows whole of right lower lobe collapsed.

birth. In this patient it seems probable that the condition is not one of congenital atelectasis, for the following reasons :—

(1) The right lower lobe, as represented by the triangular shadow in the skiagram, is larger than might be expected if it had never expanded, since under the latter circumstances it would be unlikely to grow with the child.

(2) The extreme displacement of heart and trachea to the right indicates both collapse and fibrosis rather than congenital atelectasis. In congenital cases, expansion of the upper and middle lobes should fully compensate for atelectasis of the lower lobe.

(3) The division of the main bronchus shows that the collapsed area represents the whole of the right lower lobe and gives no evidence of an accessory lobe or other congenital abnormality.

(4) In the majority of these cases, there is a definite history of symptoms dating from pneumonia or measles in infancy or early childhood, and though in this case symptoms have been minimal, the illness at 18 months of age suggests the possible origin of the collapse.

The term " collapse " bronchiectasis has therefore been used in preference to that of " atelectatic bronchiectasis," although the latter is generally taken to include both types of case.

With regard to treatment, carbon dioxide inhalation had been ineffective to expand the collapsed lobe, and the views of members on the advisability of lobectomy would be welcomed.

Discussion.—Dr. PHILIP ELLMAN said that the bulk of these cases of saccular bronchiectasis were congenital in origin, and there was no reason to suppose that this case was an exception to the rule. He thought it might be regarded as a case of congenital atelectatic bronchiectasis.

He thought that, since the ordinary palliative measures did not appear to have been of much assistance, and since this was a case of bronchiectasis limited entirely to one lobe, the question of lobectomy should be given serious consideration. If much time was lost there was a risk of a spill-over and spread into other portions of the lung; it would then be too late for lobectomy. In the hands of an expert thoracic surgeon—and this must be emphasized—this operation, certainly a major one, was recognized as the only real method of radically treating the disease, and here was an excellent indication for it.

Dr. J. L. LIVINGSTONE said that in his opinion operation at the moment was not justified. The danger would come at puberty, when the chest enlarged and more definite bronchiectasis was likely.

He thought the child should be watched carefully with regard to displacement of the mediastinum ; when that began to show there would time enough for phrenic avulsion or lobectomy.

Spondylitis Adolescens treated Three Years Ago by Wide Field X-ray Therapy of Low Intensity.—S. GILBERT SCOTT, D.M.R.E.

This patient was first seen at the British Red Cross Clinic for Rheumatism at Dr. Nissé's request, in September 1931, when she was 23 years old.

Clinical diagnosis.—Typical spondylitis deformans.

Radiological diagnosis.—Spondylitis adolescens with active infection of both sacro-iliac joints. Early calcification of spinal ligaments (lumbar).

Condition, 1931.—Whole spine fixed and tender. Unable to bend or walk. " Waddling " gait. General condition poor. Losing weight.

Past history.—At 17 years of age, prespondylitic syndrome—namely, periodic attacks of so-called muscular rheumatism of various parts of the body. At 21 years of age, severe pain of right thigh radiating to knee for six weeks ; finally settled in back and spread up whole spine. Had seen thirteen specialists and doctors ; all confirmed the diagnosis and gave a bad prognosis.

Family history.—Bad. Uncle had had same complaint, and died of excessive spinal deformity at the age of 38. Several other members of family have definite rheumatic history.

Treatment.—As the sacro-iliac joints were not ankylosed but showed definite evidence of an active infective condition, it was decided to subject the patient to the wide-field radiation with X-rays of low intensity. The whole posterior surface of the body was radiated by this method. The patient had six treatments in one month, and in six weeks was able to touch her toes; finally full spinal movement was recovered. Her general health showed a remarkable improvement—she put on over a stone in weight in six months. In eight weeks she was able to dance and do mountain-climbing. She has now a single dose about every six months as a prophylactic measure.

Several other cases have been treated by the same method, and it would appear that if the infection of the sacro-iliac joints is active, response to X-rays may be expected. Deep X-ray therapy (short wave) should never be attempted.

The vanadic acid test, a serological examination, is now being tried in these cases as a guide to dosage, and appears to form a very valuable adjunct to X-ray therapy generally.

Discussion.—Dr. J. POYNTON said that the sedimentation rate, though frequently raised in rheumatoid arthritis, did not reach such heights (e.g. 70) as in acute rheumatism. Usually 20-40 was found. Did Dr. Gilbert Scott differentiate a special form of arthritis deformans in which the first lesion was that of the sacro-iliac joints? If so, this was an interesting and important clinical fact for further investigation.

Dr. G. SCOTT (in reply) said that he did not differentiate a special form of arthritis deformans, but had found that all cases of spondylitis deformans in the young patient commenced with a lesion of the sacro-iliac joints.

Aortic Disease of Unusual Evolution.—COURTENAY EVANS, M.D.

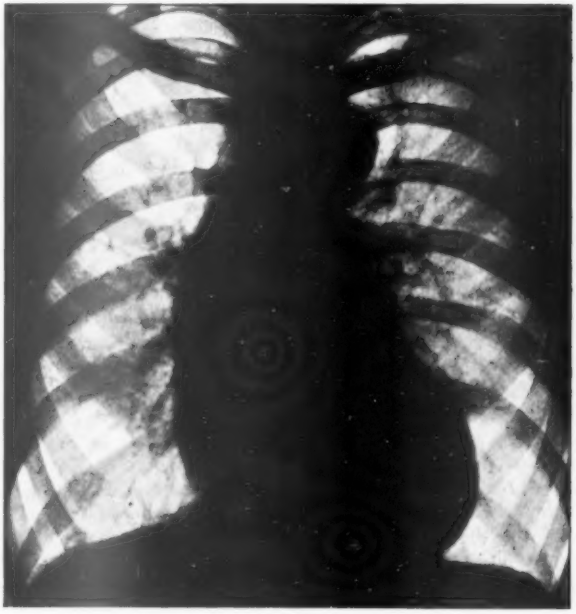
J. C., male, aged 50, general labourer in a gas company. In August 1932, became short of breath, easily tired, and later pale. On admission to St. Bartholomew's Hospital in January 1933, he was found to have a "secondary" type of anæmia. Hb. 40%; R.B.C. 5,000,000; W.B.C. 10,400; polys. 66%; lymphos. 30%; eosinos. 2%; monos. 2%. Sputum negative for tubercle bacilli. Wassermann reaction negative. Radiograms of chest showed a heart in which the right side was slightly enlarged and the aortic knuckle prominent; an old apical scar (left) and an old dense focus at the right lower zone. Heart clinically normal. Blood-pressure 108/70. No cause for anæmia found; pyrexia for eight days during stay in hospital. Great improvement with iron. Returned to work April 1933.

Quite fit till March 1934. While away from work on account of gastric trouble, experienced a sudden attack of loss of speech, paralysis of the left side and unconsciousness lasting two or three seconds. Later there were several attacks of pain between the shoulders and over the heart, passing down both arms; the pain came on during rest or on exercise, and sometimes lasted an hour.

Readmitted to hospital in May, when the heart was found to be enlarged and the impulse heaving. There was some pulsation and a systolic thrill in the second right intercostal space and in the suprasternal notch. Systolic and diastolic murmurs at apex and base. Pulse collapsing in type, the right weaker than the left and showing a definite lag. Blood-pressure, right 112/40; left 124/40. Very slight clubbing of fingers. Examination of central nervous system showed an absent left ankle-jerk. Blood-culture negative. Wassermann reaction (blood) persistently negative. The urine showed a very occasional red cell; no casts seen. Blood-count: Hb. 94%; R.B.C. 5,780,000; W.B.C. 12,000. *Differential*: Polys. 56%; lymphos. 40%; large monos. 1·5%; eosinos. 2%; basos. 0·5%. Skiagrams of heart: shadow

wider than in 1933. Report on aorta (Dr. J. B. Sparks): "The descending portion takes a sudden curve to the right, so that it projects more than 3 cm. to the right of the mid-line; ascending aorta more prominent than in 1933."

Electrocardiograms show a progressive flattening of the T waves and the development of left axis deviation. Brachial artery tracings show a lag of 0·15 second of arterial wave of right artery after left.



Skiagram of chest, June 1934.

In hospital for two months; one pyrexial attack, T. 100·6° for a few hours. Progress: Pain in the arms and chest still occurs. Treated with mercury inunctions and novarsenobillon injections.

I am indebted to Lord Horder for permission to show this case.

Discussion.—Dr. POYNTON asked if there had been any factor in the daily employment which might have caused a rupture of the aortic valve?

Dr. COURTENAY EVANS (in reply) said that there was no reason to imagine that the patient's recent occupation was either unduly arduous or likely to cause a rupture of the valve.

? Generalized Osteitis Fibrosa. Case for Diagnosis—ERNEST FLETCHER, M.B.

J. W., male, aged 36, admitted to hospital on 24.3.34, complaining of pain in both hips and thighs on walking.

Previous history.—In September 1931, patient was kicked while playing football, and had pain in the left leg for two months. He then had pain in the right foot which went up the thighs; and he now has pain in both hips and thighs. The legs

feel weak and he cannot walk without sticks, but his real difficulty is that walking is painful.

Family history.—Nothing of interest.

Condition on examination.—General condition is fair; there has been no pyrexia; average pulse-rate 84. Central nervous system, chest, and abdomen are normal. Clubbing of fingers present.

Hip-joints: neither hip-joint will fully extend; there is always 10° flexion. Flexion is full on the left, almost full on the right.

Unable to abduct the thighs. Cannot cross the left leg: the right leg can be crossed about 10°.

Gait: Shuffles along a few inches at a time and complains of much pain.

Special investigations.—Urine: Specific gravity varies from 1020-1030. Reaction acid. Albumin: a trace. Sugar, + +. Acetone present.

Sugar tolerance curve: Fasting sugar 0·084%. The curve is normal, returning to 0·084% in two and a quarter hours, always with sugar and acetone in the urine.

Blood-count: R.B.C. 5,500,000 per c.mm.; Hb. 112%; C.I. 1·0: W.B.C. 9,000 per c.mm.; polys. 76%; lymphos. 21%; large monos. 2%; eosinos. 1%.

Blood sedimentation rate (Westergren): At one hour, 2 mm.; at twenty-four hours, 24 mm. Blood-calcium, 11·35 mgm. per 100 c.c.

Wassermann reaction: Blood, + +; cerebrospinal fluid, negative.

Cerebrospinal fluid: Normal.

Radiology: The whole skeleton was examined by X-rays and the following abnormalities detected: *Pelvis and hip-joints*: Heads of both femora flattened. The left one shows a slipped epiphysis. The horizontal ramus of the left pubic bone shows an area of destruction, and there is a similar area in the upper part of the right femur, as also in the transverse processes of some of the lumbar vertebræ. *Skull*: The bones present a somewhat woolly appearance, but no areas of destruction are seen. *Hands*: The left hand shows a translucent area at the base of the second metacarpal bone, and the bones generally show some atrophy.

The blood phosphorus has not been estimated.

Discussion.—Dr. F. PARKES WEBER said that the possibility of the case being an atypical one of Recklinghausen's generalized osteitis fibrosa had not been excluded. This involved the question of parathyroid adenoma.

Dr. GILBERT SCOTT said that the general decalcification of bones of the skeleton, especially noticeable in the radiograph of the hand, clearly indicated a metabolic disturbance and suggested the possibility of some pathological condition of the parathyroid, but not necessarily a tumour, as this gave rise to a fibro-cystic condition of the bones, in addition to decalcification. There were no bone-cysts in this case.

Dr. POYNTON said he wondered whether this was a case of rheumatoid arthritis, or, rather, of arthritis deformans with a syphilitic basis. These cases had been described, particularly in France by Dufour.

Chronic Ulcers on the Soles of the Feet, of Unknown Pathology.—H. J. B. ATKINS, F.R.C.S. (introduced by the PRESIDENT).

G. E., aged 23, a waiter, complains of persistent ulceration of the sole of the left foot, the right foot having been amputated on account of a similar condition a year ago. No previous history of importance.

Family history.—A sister died of phthisis.

History.—A callosity under the head of the first metatarsal of the right foot developed what was described as a "red scab" which, with the application of hot

fomentations, broke down to form an ulcer. To relieve the pressure the patient resorted to walking on the outside of his foot, and shortly afterwards another ulcer appeared under the head of the fourth right metatarsal. The ulcers were curetted and under treatment by rest in bed tended to disappear, but broke down again as soon as the patient got on his feet.

X-ray examination 1931: Bony changes in head of fourth metatarsal in relation to ulcer. This was again scraped; at the same time tenotomy was performed in order to relieve hyperflexion of right toes. Complete healing followed after two months' rest in bed and the application of ultra-violet light, but in 1932, when work was resumed, the leg became swollen and blue, and in two months the ulcers had recurred. A further period of rest and light treatment caused the ulcers to heal, but again they reappeared on walking. With each recrudescence there was a painful swelling in the right groin. No tingling or numbness; no apparent weakness of the limb, and no disturbance of micturition at any time.

1933: Admitted to Guy's Hospital under Dr. C. P. Symonds. On examination: Ulcer about the size of a shilling under right metatarsal head, with necrotic semi-liquid centre; similar ulcer, somewhat smaller in size, under head of fourth metatarsal of same foot. Pulsation in dorsalis pedis artery present. Some wasting of thigh, and pes cavus and hammer toe on right side.

Examination of central nervous system showed (1) high degree of myopia; (2) absent ankle-jerks; (3) ? area of undissociated anæsthesia of both legs, corresponding to distribution of L.5 and S.1; (4) loss of sense of position of right big toe. Wassermann reaction negative; cerebrospinal fluid normal. Suggested diagnosis of spina bifida occulta negatived by X-ray examination. All other systems normal.

March 1933: Symes' operation performed by Mr. Lancelot Bromley; healing was good.

October 1933: A similar ulceration appeared at the base of the first left metatarsal. Eusol dressings applied locally; no improvement to September 1934. Readmitted to hospital. Recent investigations of blood and cerebrospinal fluid confirm previous findings.

(I am indebted to Mr. E. C. Hughes for permission to show this case.)

Erythro-leukæmia, in which the Myeloid Leukæmic Component is, as usual, of the Benignant Type—F. PARKES WEBER, M.D.

All the various cases of erythræmia and erythro-leukæmia can be arranged as points corresponding to the links in a chain uniting simple erythræmia (in which the erythocytes, but not the myelocytes, are increased in number) with the exceedingly rare cases in which undoubted malignant myeloid leukæmia has supervened. The majority of old cases of splenomegalic polycythæmia of the Vaquez-Osler type show a greater *relative* increase of granulocytes than of erythrocytes and are really cases of erythræmia associated with a non-malignant type of leukæmia, in which very few of the granulocytes are unripe. This type of leukæmia is of extreme rarity when not associated with erythræmia. In very rare cases the leukæmic component of the erythro-leukæmic syndrome becomes malignant, and the granulocytes are enormously increased in number and include a considerable proportion of unripe forms (myelocytes), as in ordinary cases of (malignant) myeloid leukæmia. In the present case the leucocytes mostly vary between 20,000 and 30,000 per c.mm. of blood, and are mainly polymorphonuclear neutrophils; the leukæmic component of the erythro-leukæmic syndrome is therefore, as usual, of the non-malignant type; in fact, it is no more malignant than the erythræmic component is.

The patient, Mrs. R. B., now aged 55 years, was first shown before this Section on March 11, 1932, as a case of "Erythræmia or Erythro-leukæmia," and for the findings at that time and the previous history I must refer to the published account, *Proc. Roy. Soc. Med.*, 1932, xxv, p. 964. Since then the symptoms and general condition, though varying from time to time, partly in connexion with treatment, have remained, on the whole, much the same. The last blood-count (September 27, 1934) was: Hæmoglobin 116%; erythrocytes 8,800,000, leucocytes 24,250. The treatment has included the cautious use of phenylhydrazin, occasional blood-letting, splenic treatment (by intramuscular injections of extract. lienis-Organon) and a trial of excess fat in the diet (somewhat after H. Rothmann, *Zeitschr. f. klin. Med.*, 1933, cxxiii, p. 620). On the whole the treatment seems to have been beneficial, excepting perhaps the fatty diet, which was soon discontinued, as the patient thought it made her feel ill.

For further explanation on the main points in the classification of cases of erythræmia and erythro-leukæmia, and their nature as representing "neoplastic mutations" in the bone-marrow, *see* F. P. Weber, *Med. Press*, 1929, clxxix, p. 475, and 1934, clxxxviii, p. 286.

Dr. J. GRAY CLEGG said that he had only seen one case of Vaquez's disease before. He was called into private consultation with regard to an eye which was affected with slight iritis, with haze of the media. On October 13, 1926: Dr. G. E. Loveday had reported "Hb. 134%; C.I. 0·67; R.B.C. 9,860,000; W.B.C. 17,400. *Differential*: Polys. 83·8%; lymphos. 9·2%; large monos. 2·0%; eosinos. 2·6%; mast cells 2·4%. There was inequality of size in the red cells and some shadow forms were seen. Blood-platelets were numerous. The leucocytes were mature and some of the mast cells showed the large granules noted on a previous occasion."

In a number of these cases very marked engorgement of the retinal veins occurred, but the arteries showed no change. In the present case there was merely fullness of the retinal veins and an entire absence of the hæmorrhages sometimes associated with venous engorgement.

Recent Ulnar Nerve Palsy associated with an old injury to the Elbow-joint and a Cervical Rib.—H. L. C. WOOD, M.S.

Female, aged 57. Fracture of external condyle of humerus in childhood, with marked cubitus valgus. Wasting of small muscles of hand, of recent onset. Electrical reactions suggest ulnar involvement only.

Is the present lesion due to the cervical rib or to involvement of the ulnar nerve at the elbow-joint?

Chronic Arsenical Poisoning. ? Carcinoma of the Liver. — C. E. NEWMAN, M.D.

George M., aged 35, has had œdema of the legs for four months—since just before an operation for hernia. Weakness, flatulence, and abdominal disturbance with definite pain. Appetite poor; thirst. Frequency of micturition without polyuria. Jaundice for three months.

Past history of amœbic dysentery in 1919. Has taken six minims of liquor arsenicalis three times a day since 1918 on account of dermatitis herpetiformis and now shows typical arsenical pigmentation but no neuritis. Had a slightly silvery tongue when first seen.

Has now: Œdema of the legs and abdomen; ascites with 0·3% of protein in the fluid; veins visible on chest but not on abdomen; obstructive jaundice; a very large

firm, square-shaped liver with two swellings on it—not tender; slight pyrexia and tachycardia.

Blood-count: R.B.C. 3,500,000; C.I. 1·11; W.B.C. 6,200. *Differential*: Polys. 80·4%; lymphos. 13·6%; eosinos. 5·2%; hyals. 0·1%.

Hijmans van den Bergh: cholebilirubin 7·55 mgm. %; hæmobilirubin 1·25 mgm. %.

Cirrhosis of the Liver. ? Congenital Syphilis.—C. E. NEWMAN, M.D.

Gladys C., aged 8 years.

Family history.—Patient is one of six children: (1) Boy born in 1919, alive and well; (2) girl, died of sarcoma of tonsil at age of 2½ years; the mother's Wassermann reaction was, at that time, negative; (3) and (4) twins, born in 1925: girl now alive and well; boy, stillborn; (5) The patient, born in 1926. In 1927 the mother had a miscarriage; (6) girl, born in 1931, alive and well.

History of present condition.—Patient was well until June 1933, when she had an attack of vomiting and anorexia followed by jaundice, with pale stools and dark urine. The spleen and liver were found to be enlarged, as they are now. Patient was admitted to the Belgrave Hospital. The jaundice gradually decreased but never completely disappeared. In April 1934, ascites developed and there was a return of the jaundice and itching. Patient was readmitted to hospital; she was repeatedly tapped and she gradually recovered. Pyrexia was present only after the tappings. Van den Bergh reaction: cholebilirubin, 1·7 mgm.%; hæmobilirubin, 0·55 mgm.%. Ascitic fluid, 1·2% protein.

The child remained well until September 1934, when she was again admitted to hospital on account of the return of jaundice and ascites. Her Wassermann reaction was positive in 1933. In subsequent testing it has twice been positive and three times negative. The mother's Wassermann reaction has been tested three times and was negative throughout.

Diabetes and Pulmonary Tuberculosis treated by Artificial Pneumothorax.—J. L. LIVINGSTONE, M.D.

Mrs. A. G., aged 31.

History.—October 1932: Diabetic symptoms first noticed. November 1932: Diabetic condition investigated by Dr. R. D. Lawrence and controlled with 42 units of insulin daily, carbohydrate 150 grm. December 1932: Found to have fairly extensive pulmonary tuberculosis of exudative type throughout left lung. Temperature rose to 102° F. Sputum positive. Weight 7 st. 7 lb. December 30, 1932: Left artificial pneumothorax induced; fair collapse obtained. Temperature settled in ten days.

May 6, 1933: Discharged from King's College Hospital. No cough, no sputum. Weight 7 st. 8½ lb. Blood sedimentation rate (Westergren) 13·5 mm. Insulin, 30 units. Carbohydrate, 150 grm. Skiagram showed satisfactory collapse of left lung, with apical adhesions. Refills every three or four weeks.

March 1934: Effusion into left pleural cavity with fever for six weeks. Blood sedimentation rate 63 mm. April 1934: Left lung expanding by pleuritis and pushing fluid up towards apex. September 1934: Refills becoming difficult. Insulin 22 units. Blood sedimentation rate 13 mm. Gas replacement to show the position of apex of lung which was found to be expanding. Refills stopped. Weight 8 st. 6½ lb. October 2, 1934: Left phrenic avulsion by Mr. J. B. Hunter. Insulin 22 units. Blood sedimentation rate 13 mm. Clinically very well.

An Early Case of Spondylitis Adolescens at present under Treatment by Scott's Method.—F. HERNAMAN-JOHNSON, M.D.

Ronald B., aged 21. Condition diagnosed in Orthopædic Department, Croydon General Hospital, as spondylitis (Marie-Strümpell type).

History.—Eighteen months ago began to have pain in sacral region; this extended to sides. Much pain at nights. Could barely walk 100 yards. X-ray examination showed active infection of sacro-iliac joints.

Treatment.—Wide-field X-ray treatment begun May 18, 1934; twenty-four sittings to whole of spine, but mainly to lumbo-sacral regions.

Result.—Patient has now very little pain; can walk 3 or 4 miles.

Section of Dermatology

President—HENRY MACCORMAC, C.B.E., M.D.

[*October* 18, 1934]

Two Cases of Lupus Erythematosus treated with Stovarsol.—H. MACCORMAC, C.B.E., M.D. (President).

Stovarsol is a pentavalent arsenical compound and was among those considered and discarded by Ehrlich in his classical researches. It was reintroduced by Levaditi and is largely used in the treatment of amœbic dysentery, malaria, and yaws.

A paper by R. Volk appeared in the *Wiener klinische Wochenschrift* xlvi (i), 732, and was summarized in the *Medical Annual* of 1934, by Dr. A. M. H. Gray. There Volk refers to the use of spirocid, the equivalent of stovarsol, in lupus erythematosus. Following Volk, I have used stovarsol in three cases, with marked benefit in two.

I.—Miss E. B., a hospital nurse aged 35, first developed lupus erythematosus on the nose thirteen years ago. Her mother died from pulmonary tuberculosis. About seven years ago symmetrical patches appeared at the sides of the upper lip. She then had a series of bismuth injections intermittently for five years, without benefit. Two years ago she came under my care, when she was given four injections of sanocrysin followed by marked improvement, which, however, only lasted six weeks. Subsequently T.A.B. injections and local applications of carbon dioxide were tried but there was no improvement. In June 1934 two tablets of stovarsol (each 4 gr.) were given by mouth daily for one month. At the end of this period acute arsenical dermatitis developed, with an extensive morbilliform eruption and pronounced œdema of the face and legs. Patient was taken into hospital and treated by intravenous sodium thiosulphate; she made a complete recovery. As the lupus erythematosus had not completely subsided, stovarsol was again given but immediately discontinued as it again produced an erythema. The eruption, however, has continued to diminish and is now at least temporarily cured.

II.—Miss D. M., aged 27, first observed the eruption on the right cheek ten years ago. It continued to develop, gradually involving the whole face, the exposed V-shaped area on the chest, and the hands. She had been treated by krysolgan in 1927, and subsequently by bismuth, without effect. In July 1934 she was admitted to hospital and treated by stovarsol, two tablets daily for one week, when an extensive erythema developed. After one month when the erythema had completely

cleared up, stovarsol was again tried and it was found that one tablet every other day could be taken without symptoms of intoxication appearing. The eruption has steadily decreased and it seems likely that it will be completely effaced.

It will be noted that in both these cases the improvement followed a severe arsenical reaction. I have also observed, when giving gold salts, that the most consistently good results have been obtained when some degree of reaction followed the gold injections and it seems that in some part the improvement in these two cases should be attributed to this.

Discussion.—Dr. H. W. ALLEN asked whether any member had seen cases of generalized dermatitis following treatment by stovarsol. He had seen one such case.

Dr. H. SEMON referred to a case which he had published and described with a short analysis of the literature.[1] The patient, a married woman, aged 32, had been suddenly taken ill with a rigor on July 9, with a rise of temperature to 100·6 on July 10, a morbilliform and punctate eruption appeared on the face and neck, and later on the arms, hands, back and trunk during the afternoon and evening of that and the ensuing day. The patient's sister, with whom she had been in contact ten days previously, was suffering from German measles, and although the incubation period of that disease is in the neighbourhood of seventeen days this diagnosis had been suggested. The eruption would certainly have passed criticism, but in addition to the discrepancy of dates, there was the further symptom of repeated attacks of burning and tingling of the extremities, and on perusing the nurse's notes one found the significant fact that a tablet of stovarsol had been administered on July 9, 12, and 14.

Further inquiry elicited the information that the patient was under treatment for chronic mucous colitis and had been given the drug for a considerable time (40 gr. in all). The eruption and subjective symptoms subsided promptly on its being withdrawn and substituted by calcium-urea (afenile), and solutions of strontium bromide (eczebrol), intravenously. He (Dr. Semon) agreed that the effect of the stovarsol in one of the two cases exhibited might have been due to the dermatitis which had been provoked. He remembered a case of his own—an exfoliative dermatitis of very severe degree following an excessive injection of a gold salt, in which subsequent to its clearance after six months in hospital, the chronic small patch of lupus erythematosus for which it had been administered temporarily disappeared.

Dr. P. B. MUMFORD agreed with what had been said as to the effects of a violent reaction. In one of his cases with an extensive lupus erythematosus eruption a severe reaction developed after 2 grm. of myocrisin. The areas of lupus erythematosus on the scalp became exudative and the face became œdematous within twenty-four hours. The patient returned the following week much improved. Three weeks later the lupus erythematosus had almost disappeared.

Dr. HUGH GORDON said that he had known one case of severe arsenical dermatitis following upon the use of stovarsol; the drug had, however, been used continuously over a period of three months. He had never heard of dermatitis supervening after such small dosages as in the cases under discussion.

He had recently used stovarsol in a group of cases of severe alopecia areata in children where syphilis was suspected. He had employed the dosage as recommended by Oppenheim, i.e. two to three tablets, each of 4 gr., to be given with water first thing in the morning on an empty stomach for three days, followed by a rest interval of three days. The total dosage per course should consist of as many 4-grain tablets as the patient's weight in kilos.

He had not, as yet, observed reactions of any kind.

Dr. G. B. DOWLING said that the observation that had been quoted bore out Dr. Lyle Cumming's contention, that gold acted not on the tubercle bacillus directly but as a chemical irritant on the chronic inflammatory lesion. It seemed that it had a similar action on chronic lupus erythematosus; it was necessary to stir it up before it would resolve.

[1] Acute stovarsol dermatitis, *Lancet*, 1932 (ii), 340.

The PRESIDENT (in reply) said that in his two cases a severe toxic erythema, without exfoliation, was observed. While he considered that this reaction had exerted a beneficial effect, account must also be taken of the therapeutic qualities of the drug itself.

Professor Cumming's experiments seemed to require a different interpretation. When gold salts and tubercle bacilli were injected simultaneously, guinea-pigs developed tuberculosis, but if the gold salts were withheld until later, protection was obtained. A parallel to this had been noted in syphilis; observations in France and Belgium had shown that prophylactic salvarsan did nothing except interfere with and delay the course of early syphilis. In short it seemed essential for the infection, whether tuberculous or syphilitic, to become "fixed" before either gold or arsenic could exert their specific action, implying that these salts acted indirectly by stimulating the natural protective substances.

Pemphigus treated with Germanin.—G. B. DOWLING, M.D.

Mrs. M., aged 66, developed a bullous eruption which quickly became universal about March or April 1934. Admitted to St. Thomas's Hospital May 22, and discharged on August 24.

Condition on admission.—The patient's skin was covered with bullæ, large and small, some of which had become confluent with others and after rupturing left large raw areas. In addition there were shallow ulcers in the mouth and nose.

There was no eosinophilia evident in the blood or in the blister fluid, which was sterile on culture.

Treatment.—Germanin was given in doses of 0·2 grm. to 0·5 grm.—three doses a week at intervals of a week or longer. Improvement began after about the fourth dose and has been maintained until the eruption cleared up completely.

Although there has has been no return of the bullæ there has been a considerable loss of weight and persistent loss of appetite.

Discussion.—Dr. H. W. BARBER said that in Germany[1] the opinion seemed to be that, although the effects of germanin in the treatment of pemphigus and dermatitis herpetiformis might be favourable, relapse was likely to occur when the treatment was discontinued.

The PRESIDENT said that at a meeting of the Section he had shown a case of pemphigus vegetans which had been treated with germanin and radium. After the radium treatment a chronic sinus had developed but had now cleared up and the blister formation had entirely ceased, and the disease seemed to be cured. He had not had success in any of the other cases in which he had tried germanin.

Dr. I. MUENDE said that about a year ago a paper had been published[2] relating cases of pemphigus and dermatitis herpetiformis treated with germanin. Two of these cases relapsed and eventually died, and the author of the paper suggested that death was probably precipitated by the second treatment with germanin, and concluded by regarding germanin as a rather dangerous drug. A similar observation was made by Bode.[3]

Dr. L. FORMAN supported Dr. Muende's remarks. He had under treatment a patient with extensive pemphigus vegetans, who had done well under treatment by rest in bed and arsenic. The vegetating lesions had flattened out and blisters had ceased to appear. Germanin was given in doses recommended by the makers. The temperature went up and the bullæ reappeared in large numbers, and there was deterioration of the general condition. He felt that it was necessary to use the drug with considerable caution.

Dr. DOWLING (in reply) said that in this case at any rate one must attribute the result to the use of germanin, and it was at least possible that the patient would have continued to suffer a great deal if it had not been used.

[1] Zoon, J. J., *Acta dermat-venereol*, 1933, xiv, 473.

[2] Id.

[3] *Dermat. Wochenschr.*, 1933, 1160.

Myxomatous Degeneration associated with Hyperthyroidism.—G. B. DOWLING, M.D.

The patient, a man aged 63, has been a planter in South America. About three years ago he developed hyperthyroidism and at about the same time symmetrical infiltrations appeared on both legs. Originally these were sharply defined, encircling

FIG. 1.

both legs in their lower two-thirds. The surface was mammilated, with well-defined lumps in places (fig. 1).

Following an operation of partial thyroidectomy in August 1933, the areas have gradually become almost flat and the rugosity has quite disappeared. However, about half a dozen new circumscribed infiltrations have been appearing gradually during the past six months.

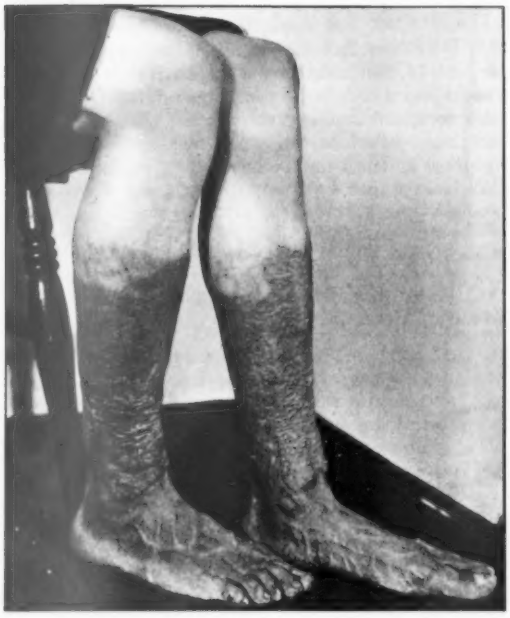


FIG. 2.

The case is exactly similar to that of a man aged 24, whom I showed at the meeting of March 15 of this year[1] (fig. 2).

Morphœa with Spontaneous Resolution.—J. H. T. DAVIES, M.B.

Mrs. F. B., aged 24; two children (girls) both healthy. Father died, aged 52, from tuberculosis; mother died, aged 54, from carcinoma of cervix. She has one brother mentally defective from meningitis in infancy. She is healthy herself; her history consists of only minor childish ailments and operations for appendicitis and hernia.

The condition began near the left breast when she was 13 years old. She describes the patches as starting as little white hard lumps like insect bites, itching at first, but not inflamed or reddened in any way. The lumps multiply for a while and then gradually disappear again leaving a pigmented stain. The eruption consists for the greater part of brownish stains; the edges of the older lesions fade insensibly into the surrounding skin, but are more distinct in other parts. The skin in these brownish patches is nearly unaltered in texture. It is evidently thinner than normal because superficial veins show through it and a faint capillary network is visible. At the edges of some and in the middle of others of these patches can be seen traces of a scleroderma-like process; but instead of the usual dense smooth white plaques, the thickening is broken up into nodules and ridges. Some of the nodules are inconspicuous and are scattered diffusely over areas other-

[1] *Proceedings*, xxvii, 1934, 1361 (Sect. Derm., 63).

wise apparently completely resolved. On the back of the left shoulder is an example of another type of lesion, but the scleroderma-like process is absent or transitory and the normal skin is broken up into islands by irregular areas of atrophy.

Microscopical report.—" Section shows large masses of acellular collagen in the sub-epithelial tissue. There is a little round-celled infiltration around some of the dilated vessels. Appropriate staining shows the elastic fibres in the corium still present. The overlying epithelium shows a tendency for the papillæ to disappear. Much pigment is present in the basal cells. " It is evident that we are dealing with a scleroderma in the later stages."

Dr. A. M. H. GRAY said that he thought massage was of considerable help in clearing up these cases, whether of the diffuse or the localized type.

Pigmented Mole of Foot.—J. H. T. DAVIES, M.B.

This patient, a girl, aged 18, has a pigmented warty mole occupying the first four toes and interdigital spaces of the right foot. Between the toes the surface is thrown into ridges and folds which become filled with débris resulting from maceration and infection of the surface.

In the case of a mole occupying the flat surface on the feet or elsewhere, the question, so far as I am concerned would not arise, but in the present instance I feel that there is a distinct opportunity for nævo-carcinoma to develop, as the result of irritation. Amputation, however, would mean a grave disability, and I am anxious to obtain the advice of the Section in order to decide whether to remove the risk of nævocarcinoma at the expense of this disability, or to let the patient take this risk and retain what is otherwise a perfectly useful foot.

Discussion.—Dr. A. M. H. GRAY said that if it were considered desirable to carry out any surgical procedure in this case—and he considered it was—the whole thing should be taken away. He did not favour any plastic manœuvres, especially as grafted skin seemed to be more susceptible to damage than other tissue.

Dr. I. MUENDE said that this case was of the papilliferous type in which the pigmentation was not greatly marked, and was confined chiefly to the nævus-cells in the corium. The variety which he thought was considered by most authorities to-day as being more prone to malignant change was the slaty-gray or black type, in which considerable pigmentation was to be found in the separated lower poles of the rete pegs. He thought that the prognosis in the present case was good and would prefer to leave the growth alone. His view was supported by examination of tissue from such cases.

The PRESIDENT said that Dr. Davies stressed the septic infection between the toes as a source of chronic irritation which might in time provoke malignant degeneration. Apart from this the pigmented mole presented no especially sinister qualities. He (the President) thought, therefore, that before any more radical procedure was undertaken an attempt should be made to correct the sepsis.

? Lichen Planus in an Infant.—J. H. T. DAVIES, M.B.

When this child was born in the Sussex Maternity Hospital nine weeks ago no skin lesion was noticed. Within a few days, however, a rash appeared and covered the trunk, face and extremities. It was described by the house-surgeon as a " copper-coloured papular rash," and syphilis being suspected, Wassermann tests were carried out on mother and child, in each with negative results. At seven weeks I saw the child. There was no sign of widespread eruption, but on the hands and feet were linear hyperkeratotic and warty streaks resembling nævus unius lateris with red infiltrated base. The eruption was present also on the wrists, forearms, and ankles,

mainly in the form of a retiform arrangement of papules isolated or confluent into linear forms. In addition to the lesions on the hands and feet, which have somewhat diminished under treatment (zinc ointment with vaseline), there is a generalized rash of erythematous papules. The mother states that it differs from the original rash which seems to have been more solidly papular.

The parents are healthy, and there is no family history of skin disease. This is the first child. She has been well, except for the eruption, since birth and has progressed normally. She has never had any soothing powders or other drugs.

As regards the diagnosis, I searched carefully for a typical lichen planus papule without success. The linear and retiform distribution and the colour of some of the lesions suggested lichen planus to me but I should be happier with an alternative diagnosis.

Discussion.—Dr. DOWLING said that he regarded the case as one of linear nævus.

Dr. R. KLABER said he considered that the condition was lichen planus.

The PRESIDENT said he would have no hesitation in considering this a linear nævus with concurrent lichen urticatus. In lichen urticatus lesions indistinguishable from those of lichen planus were sometimes present.

? Acro-dermatitis Continua (Hallopeau) Secondary to Sycosis and Cellulitis.—ROBERT KLABER, M.D.

This patient, a man aged 51, has seen many years' military service in India and tropical Africa and is now occupied as a plumber. He enjoys good general health.

Two and a half years ago there was some purple swelling of the nose which spread on to both lower lids and cheeks. Soon after, this largely disappeared, but a pimple which was present just below the right commissure of the lips spread, forming a patch of vegetative dermatitis. About one year ago a diffuse, ill-defined swelling appeared on the lower part of the right cheek, and has persisted since. Six months ago a few pustules appeared on the inner side of the right big toe nail This condition spread round this toe and soon involved all the other toes of this foot and then, later, all the toes of the left foot. Subsequently all the finger nails became similarly involved until only the two outer fingers of the left hand remained unaffected. The initial pustular lesions have gone on to form on each digit a well-marked diffuse paronychial swelling with glazed red covering skin. The nails of all the affected digits have been shed, leaving a heaped-up irregular keratotic mass covering the nail bed.

Repeated examinations for fungus have proved negative. There is no certain evidence of psoriasis elsewhere.

The Wassermann reaction is negative and the blood-count showed nothing of note. The cultures from the nail folds have given a growth of *Staphylococcus aureus* and *albus*.

The patient is edentulous, but skiagrams show a retained root in the right upper jaw.

Dr. A. M. H. GRAY said this case had several possibilities. He thought it was possible to exclude psoriasis, though he did not attach much importance to the appearance of the nail folds. It had struck him that the nails, and also the skin on the toes well below the nails, in which there were a number of small crusted lesions, were similar to those seen in gonorrhœal keratosis. If this was so the lesion on lip had no relation to the other condition but was a chronic staphylococcal infection. It was also possible that the whole condition might be one of chronic staphylococcal infection. He gathered, however, that only one Wassermann test had been made, and that was scarcely enough to exclude the possibility of syphilis. A provocative injection of salvarsan might be a wise procedure before a final decision was made.

Two Cases of Cicatricial Alopecia. ? Pseudo-pelade of Brocq.—HUGH GORDON, M.R.C.P.

I.—There is a diffuse cicatricial alopecia of the vertex of the scalp which has been noticed for some six months and is progressing.

For two years the patient, aged 45, has complained of an irritating eruption in the front of the left leg, where there are to be seen seven or eight grouped nodules with scarring in the centre, about the size of a cherry. The eruption has the appearance of a lichen obtusus. The appearance of the scalp is not typical of pseudo-pelade as described by Brocq, in that the condition is diffuse and there are no small circular areas of baldness which have coalesced leaving islands of normal hair. The skin is slightly erythematous, but no actual infection of the follicles is present, nor is there any red halo round the normal follicles.

II.—This appears to be a typical example of pseudo-pelade of Brocq. There is marked erythema of the small circular bald patches, which was noted by Brocq in the early stages of the disease.

Cases have been shown—by Sir Ernest Graham-Little and others—of cicatricial alopecia occurring with lichen spinulosus and lichen planus. In the one case shown here there is this association. It is noteworthy, however, that clinically, in this case, the typical picture of the pseudo-pelade of Brocq is not present.

The two cases have been shown side by side with the suggestion that the lichen planus group of cicatricial alopecia may only resemble true pseudo-pelade in that cicatricial alopecia exists without any evidence of a pre-existing infective process.

There is another possibility, namely, that the association of cicatricial alopecia and lichen planus may be fortuitous; lichen planus is a fairly common condition, and in my experience pseudo-pelade appears to be getting also comparatively common.

Discussion.—Dr. DOWLING said that at a meeting several years ago he had shown a case of lichen planus with horny follicular lesions and a few patches of pseudo-pelade; the patient was a girl aged about 12. Lichen planus had developed first and later pseudo-pelade appeared on the head. The patches were not preceded by horny follicular lesions on the scalp. They differed, therefore, from the cases of cicatricial alopecia shown by Sir Ernest Graham-Little. In those cases such lesions had always preceded the follicular atrophy.

Dr. J. H. T. DAVIES said that recently he had seen a case in which the whole scalp was covered by patches of cicatricial alopecia. There was a superficial resemblance to the alopecia of secondary syphilis. There were lichen spinulosus lesions on the trunk and extremities.

Dr. MUENDE said that cases recorded under the title of cicatricial alopecia associated with lichen planus were usually those in which the bald patches were bilateral, and situated in the temporal region. In Dr. Gordon's cases, however, the patches occupied the typical situation of Brocq's pseudo-pelade, over the vertex.

? Schaumann's Disease.—C. H. WHITTLE, M.D.

R. P., a widow, aged 59, attended hospital on account of swelling of the tip of the nose which she had noticed for about a year. She was first seen by me in February 1934.

There was a bluish firm bulbous swelling of the lower inch of the nose and a brick-red plaque, about an inch in diameter and covered partly by tough adherent scales, situated over the right malar prominence. The second patch resembled lupus erythematosus.

In addition to the facial lesions there were curious swellings of the hands and feet, consisting of soft, semi-fluctuant, spindle-shaped tumours in the following positions :—

Right index—proximal and middle phalanges.
„ middle „ „ „
„ little „ „ „
„ ring distal phalanx

Left thumb—distal phalanx
„ index proximal phalanx
„ middle „ „
„ ring „ „ and also the metacarpal which is large and fluctuant

Right hallux and right second toe ; left second toe. In addition the nails of the right index and right fingers, and of the left thumb and index finger show atrophic changes.

The swellings are of considerable size, bluish in colour and not tender. There are also some small nodes in the skin on the outer aspect of the right arm. The patient appears to be otherwise well.

There is a curious history in regard to tuberculosis. The patient nursed her husband through a long—and fatal—illness ; he died from tuberculosis. She had hæmoptysis within a few months of his death, but she has had no signs of gross tuberculosis since. It has been suggested to me that she is in an anergic phase of tuberculosis, and possibly that is the explanation of the negative Mantoux reaction.

Skiagrams (which are shown) show a rather diffuse rarefaction of the shafts of the bones underlying the tumours, with a blurring of the edges and a fine trabeculation which is not apparent in the unaffected bones. There is also some widening of the shafts affected. In the subterminal phalanx of the right little finger there appears to be a small cyst in the head of the bone.

The radiologist (Dr. Roberts) has not yet given a name to these changes, but they are probably of the nature of an osteofibrosis.

Kahn reaction negative. Mantoux reaction negative (0·1 c.c. of 1 : 1,000 tuberculin) ; hæmolytic streptococcal toxin reaction positive, with a flare of 1¼ in. and central wheal.

Treatment.—General and local ultra-violet irradiation. There has been some lessening of the swelling of the nose, but the hands and feet are unaffected by treatment so far.

Discussion.—Dr. L. FORMAN asked what was the strength of the tuberculin which Dr. Whittle had used. In recent communications on lupus pernio the association with tuberculosis had been emphasized. Recently in a case of lupus pernio shown by Dr. Barber there had been shown on post-mortem examination, caseous axillary and periosteal glands. The Mantoux reaction of the patient had been positive.

Dr. DOWLING said that in the case of which Dr. Forman spoke the condition was perhaps passing from sarcoid to tuberculosis, so that the tuberculin test might be expected to change from negative to positive. Schaumann said that in the cases that he was able to follow up pulmonary tuberculosis had supervened with the disappearance of the sarcoid lesions.

Dr. WHITTLE (in reply) said that the strength of the tuberculin he used was 1 : 1,000. The dose was 0·1 c.c.

Erysipeloid (Erythema serpens).—H. SEMON, M.D.

J. M., female, aged 20, domestic servant.

History.—The eruption, which is limited to the right hand (palm and fingers), is said to have begun on the radial border of the index finger, and gradually spread in the course of a fortnight to the back of the hand, causing swelling with burning and some irritation. It has now entirely disappeared from the original sites, and is seen on the palm as a slightly raised dusky red or cyanotic serpiginous ridge. The over-

lying epidermis is not vesicular or exfoliative, and neither of these symptoms have been noted at any time during the fourteen days during which the eruption has been present. Similar abortive lesions are present on the middle finger and thumb.

The general health has not been affected in any way.

The patient's occupation as a general domestic servant—among her duties being the cutting and dressing of raw meat—suggests that as the source of her infection.

The most generally accepted cause of this dermatosis which is relatively common among butchers, fishmongers and cooks, is the *Bacillus murisepticus* (R. Koch) of swine fever, which—like its better-known human prototype, the streptococcus—tends to wander and flourish in the lymphatics, a fact which may account for the peculiar configuration of the ensuing lesions. Unlike the streptococcus, however, it practically never leads to cellulitis or abscess formation, and ends usually in spontaneous resolution, two or three weeks from the date of onset.

Attempts to isolate and grow the organism from serum expressed after puncture of the most prominent portions of the eruption were unsuccessful, both aerobically and anaerobically. A comprehensive account of the infection together with a coloured illustration (closely resembling the above-reported case) will be found in Arzt and Zieler's *Die Haut und Geschlechtskrankheiten*, 1933, iii, 257.

The PRESIDENT said that as this was a condition which produced immunity, these patients had only one attack.

Section of Orthopædics

President—ALAN H. TODD, M.S.

[*October* 2, 1934]

The Treatment of Pes Cavus

PRESIDENT'S ADDRESS

By ALAN H. TODD, M.S.

ABSTRACT.—(A) Review of earlier methods: Manipulations; fasciotomy; Phelps' operation; Steindler's operation; anterior arch-plates.

(B) Reasons for their relative failure.—(1) Correction of the deformity is imperfect, and (2) as they deal with the existing deformity only, and not with its cause, the result is not permanent; relapse occurs.

(C) Evolution of the modern operation.—Two facts in connexion with the ordinary "idiopathic" type of pes cavus are constant, and therefore noteworthy, viz.: (*a*) the deformity is entirely a fore-foot deformity, consisting of dropping-down of the fore-foot, and (*b*) paralysis of the lumbrical or of the interosseous muscles is never found at operation.

This suggests that a cause for the fore-foot drop should be sought. Pes cavus never occurs in flail foot, but may develop in mild cases of paralysis of the anterior tibial (extensor) group of muscles; this suggests that in less marked cases of paresis of these muscles, pes cavus may result; in fact, this has been observed.

Finally, a case in which the legs were known to have been normal, and one was damaged, anteriorly (thereby weakening the long extensor action) resulted in the development of typical unilateral pes cavus.

The part played by the interossei and lumbrical muscles is purely passive, and results from the dropping-down of the metatarsal heads beyond their line of action. This can be demonstrated on any case in which contracture of the soft parts of the toes has not occurred; pushing-up the anterior arch brings down the toes, and vice versa.

The problem, then, seems to consist of finding a means to strengthen the relatively weak long extensors, and of giving them a stronger and more direct lifting action upon the metatarsal heads.

(D) The modern operation.—This consists of two distinct parts: (*a*) the correction of existing deformity, and (*b*) the adoption of measures to prevent recurrence of the deformity. (*a*) Mere non-selective elongation of the structures of the sole is inadequate; those on the inner side must be lengthened and flattened more than those on the outer side; therefore, the joint-capsules, fasciæ, tendon-sheaths, etc., are divided as freely as possible, by open operation, on the inner side of the foot. Steindler's section of all structures attached to the os calcis then allows the whole foot to elongate; this is followed by vigorous manipulation, and this completes stage (*a*).

(*b*) The extensor tendons are then transplanted into holes bored through the necks of the metatarsal bones (Murk Jansen's operation, modified), and are sutured, the foot being held over-corrected meanwhile.

If the toes are contracted, and the above method does not correct the deformity, arthrodesis of the proximal interphalangeal joints is performed; the fifth toe may perhaps be amputated.

(E) After-treatment and results.—Other points: The results seem to be permanent. At what age should this operation be performed? Treatment, at earlier ages. The type and degree of disability caused by pes cavus. Relief of advanced cases.

RÉSUMÉ.—(A) Revue des anciennes méthodes: Manipulations, fasciotomies, opérations de Phelps et de Steindler; support de l'arche antérieure.

(B) Explication de leur manque relatif de succès.—(1) Correction imparfaite de la déformité. (2) Comme elles n'agissent que sur la déformité existante, et pas sur sa cause, le résultat n'est pas permanent.

(C) Évolution de l'opération moderne.—Deux faits relatifs au pied creux "idiopathique" ordinaire sont constants, et méritent en conséquent d'être retenus: (*a*) la déformité n'intéresse que la partie antérieure du pied, qui s'abaisse: (*b*) à l'opération on ne trouve jamais de paralysie des muscles lombricaux ou interosseux. Ceci indique qu'il faut chercher plus loin la cause de l'abaissement de la partie antérieure du pied. Le pied creux ne se trouve jamais dans les cas de pied ballant, mais il peut exister dans les légères paralysies du groupe tibial antérieur (extenseurs), ce qui suggère que le pied creux peut survenir à la suite des parésies de ces muscles. En effet, ceci a été constaté. Finalement chez un cas où les jambes étaient normales, un pied cave unilatéral typique survint à la suite d'une blessure au côté antérieur d'une jambe, produisant un affaiblissement de l'action du long extenseur.

Le rôle des muscles interosseux et lombricaux est passif, et résulte de l'abaissement des têtes des métatarsiens au delà de la ligne normale. On peut démontrer ce fait dans tous les cas où il n'y a pas de contracture des parties molles des orteils. Si on soulève l'arche antérieure les orteils s'abaissent, et le contraire.

Il semble donc qu'il s'agit de trouver un moyen de renforcer les longs extenseurs relativement affaiblis, et de rendre plus vigoureuse et plus directe leur action soulevante sur les têtes des métatarsiens.

(D) L'opération moderne.—Celle-ci se compose de deux parties distinctes: (*a*) la correction de la déformité, et (*b*) la prévention d'une récidive.

(*a*) Le simple rallongement non sélectif des structures de la plante du pied ne suffit pas. Celles du côté interne doivent être rallongées et aplaties plus que celles du côté externe. Pour achever ce résultat on sectionne aussi les capsules des articulations, les fascies, les gaînes des tendons du côté interne du pied aussi largement que possible. La section de Steindler de toutes les structures attachées à l'os calcis permet alors au pied entier de s'allonger. Cette opération est suivie de manipulation, qui complète la première partie du traitement.

(*b*) On transplante et suture les tendons extenseurs dans des trous percés dans les cols des os métatarsiens (modification de l'opération de Murk Jansen), tenant le pied fixé en hypercorrection.

Si les orteils sont contractés et le traitement décrit ne corrige pas leur position, on fait une arthrodèse des articulations inter-phalangées proximales. Le 5e orteil peut être amputé.

(E) Résultats définitifs. Autres questions.—Les résultats semblent être permanents. A quel âge faut-il opérer? Traitement pendant le jeune âge. Type et degré de déformation causée par le pied creux. Soulagement des cas avancés.

ZUSAMMENFASSUNG.—(A) Besprechung der älteren Behandlungsmethoden: Manipulation, Fasciotomie, Operationen nach Phelps und Steindler, Platten für due vordere Fusswölbung.

(B) Ursache des relativen Misslingen dieser Methoden: (1) Unvollkommene Korrigierung der Deformität. (2) Da sie nur die existierende Deformität, und nicht ihre Ursache berücksichtigen, ist der Erfolg nicht dauerhaft.

(C) Entwicklung der modernen Operation.—Zwei Tatsachen sind bezüglich des gewöhnlichen "idiopathischen" Hohlfusses beständig, und sind deshalb bemerkenswert: (*a*) Die Deformität ist nur eine Vorderfuss Deformität, und besteht aus einem Sinken des Vorderfusses, und (*b*) man findet bei der Operation nie eine Lähmung der Mm. lumbricales oder interossei.

Dies veranlasst das Aufsuchen einer Ursache für das Sinken des Vorderfusses. Der Hohlfuss kommt nie bei Schlotterfuss vor, er kann aber in milden Fällen von Lähmung der vorderen tibialen Muskeln (Extensoren) vorkommen. Das weist darauf hin dass der Hohlfuss in den milderen Fällen von Parese dieser Muskeln entstehen kann. Solche Fälle sind, in der Tat, beobachtet worden.

Endlich, in einem Fall wo die untere Extremitäten normal waren, trat nach Schädigung an der Vorderseite des einen Beines die die Wirkung der langen Extensoren schwächte, ein typischer einseitiger Hohlfuss auf.

Die Rolle der Mm. interossei und lumbricales ist vollkommen passiv, und geht aus der Senkung der Metatarsalköpfchen aus. Das kann man in allen Fällen beweisen wo Kontraktur der Zehen nicht besteht. Das Aufheben der vorderen Fusswölbung bringt die Zehen hinunter, und umgekehrt.

Das Problem schein zu sein, eine Methode zur Verstärkung der relativ schwachen Extensoren zu finden, und ihnen eine stärkere und mehr direkte aufhebende Wirkung auf die Metatarsalköpfchen zu geben.

(D) Die moderne Operation: Diese besteht aus zwei Teilen: (*a*) die Korrektion der Deformität, und (*b*) Massnahmen gegen Rückfälle.

(*a*) Die einfache, unselektive, Verlängerung der Strukturen der Fusssohle genügt nicht; die Strukturen der inneren Seite müssen mehr verlängert und verflächt sein als die der äusseren Seite. Deshalb schneidet man auf der inneren Seite so weit wie möglich die Gelenkkapseln, Fascien, Sehnenscheiden, usw. durch. Die Steindler'sche Sektion der an dem Os calcis befestigten Strukturen ermöglicht dann die Verlängerung des ganzen Fusses. Diese Operation wird durch kräftige Manipulation gefolgt. Das erste Stadium ist so vollendet.

(*b*) Die Extensorensehnen werden jetzt in die durchbohrte Hälse der Metatarsalknochen transplantiert (Modifikation der Murk Jansenchen Operation) und genäht, bei überkorrigierter Stellung des Fusses.

Wenn Kontraktion der Zehen besteht, und diese Operation sie nicht beseitigt macht man eine Arthrodese der proximalen Interphalangealgelenke. Der V. Zehen kann man amputieren.

(E) Nachbehandlung und Dauererfolge. Andere Fragen.—Der Erfolg scheint dauerhaft zu sein. In welchem Alter soll man operieren? Behandlung im früherem Alter. Typus und Grad der durch Hohlfuss bedingten Deformität. Linderung bei vorgeschrittenen Fällen.

SOME fifteen years ago my attention was directed to the treatment of pes cavus because, as it seemed to me, the results of all the methods of treatment then in use were transient.

It was well known that the ordinary "idiopathic" form of the disease, as it occurred in children, was prone to a slow but steady and inevitable progress, although for a time it might not cause severe suffering or, indeed, any complaint at all. Some surgeons treated the existing deformity whether the patient actually complained or not; others were content to temporize until such time as pain or other disability rendered treatment imperative. The methods usually employed included systematic manual stretching, stretching on rack-splints such as the Scarpa shoe, forcible manual stretching and wrenching under anæsthesia, tenotomy of the extensor tendons, the use of supports designed to lift the anterior arch, plantar fasciotomy, Phelps' operation, and external cuneiform tarsectomies; more recently, Steindler's operation has been introduced, and has met with much favour.

None of these methods, conservative or operative, gave a lasting result. The deformity might be overcome temporarily, but sooner or later the inveterate tendency of the disease to progress asserted itself anew, and the deformity and disability recurred. In adults recurrence was slower, of course, but it took place nevertheless.

It seemed obvious that the reason for the comparative failure of these various lines of treatment must be that they lacked some essential feature; and, on considering the facts, it seemed obvious, too, that the fault lay in a failure to remedy the fundamental cause of the deformity. It was always possible to correct the visible deformity of a pes cavus; but the underlying cause, continuing to operate, sooner or later brought about relapse.

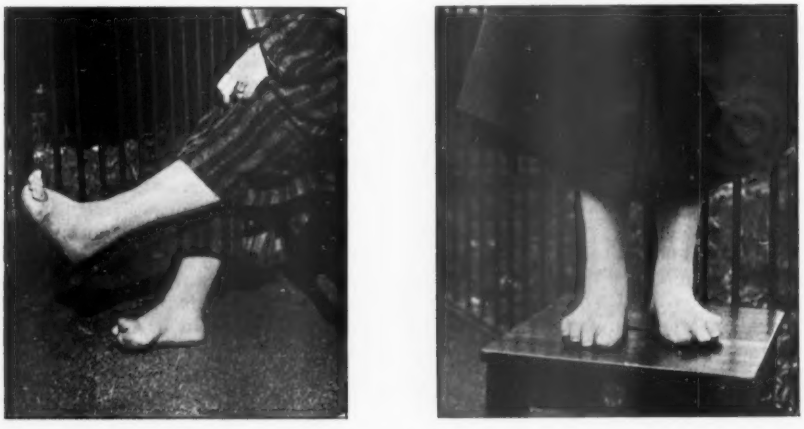
I have seen a grown-up woman (fig. 1), who underwent tenotomy of all her extensor tendons, followed by plastering, no less than four times. One admires her pluck; one marvels at the patient, dogged determination and orthodoxy of the surgeon; but surely such an experience should be interpreted as indicating that the principle of the operation was wrong and that the results of a condition were being treated, and not its cause.

The essential problem, then, seemed to be to find the cause of the deformity, and tackle that.

Two facts in connexion with the ordinary "idiopathic" type of pes cavus are constant, and are therefore noteworthy: (*a*), the deformity is entirely a fore-foot deformity, consisting essentially of dropping-down of the fore-foot; and (*b*) paralysis of the lumbrical or of the interosseous muscles is never found at operation.

Slight contracture of the tendo Achillis may be found in cases of pes cavus of many years' duration, but it is secondary, not primary; it is never present in the earlier and milder cases, and in many examples of quite severe pes cavus, the fore-foot is dropped down to the extent of an inch or more, and yet there is no shortness of the tendo Achillis at all. This is graphically illustrated, in any severe case, by covering the front part of the foot with a cloth; the leg and hind-part of the foot look perfectly normal.

Several of the leading American textbooks of orthopædic surgery state that contracture of the tendo Achillis is the fundamental cause of pes cavus. Quite apart from clinical observation, this statement seems to be manifestly wrong for another reason. It is well-known that tightness of the heel-tendon gives rise to a severe



(*a*) (*b*)

FIG. 1.—Clawed toes and pes cavus. (*a*) After repeated tenotomy of extensor tendons. (*b*) After radical operation.

form of flat foot ("valgus ex equino," or "valgus secondary to equinus"); how could a tight tendo Achillis produce two conditions so utterly opposite and different as flat foot and pes cavus?

I submit that a constant and essential feature of pes cavus is the dropping-down of the fore-foot.

Secondly, it has been stated over and over again that the lumbrical muscles, or the interossei, or both, are wasted in pes cavus, and that this accounts for the condition. No cause for this alleged wasting has been demonstrated; it has been suggested—and the remark has often been repeated—that there is some analogy between pes cavus and ulnar claw-hand. But, in fact, the alleged wasting of the interossei, or of the lumbricals, has never been seen at operation. Hundreds of open operations have been performed upon the inner arch of the foot in recent years, by many different surgeons, and the wasting in question has never yet been demonstrated. The sooner this fiction is dropped, the better.

I do not suggest that the interossei are not responsible for the production of the claw-toe element in the deformity. I think that they are, but I do most emphatically dispute the suggestion that they are ever wasted or paralysed, or that such wasting or paralysis causes pes cavus, or even clawed toes. I believe that the

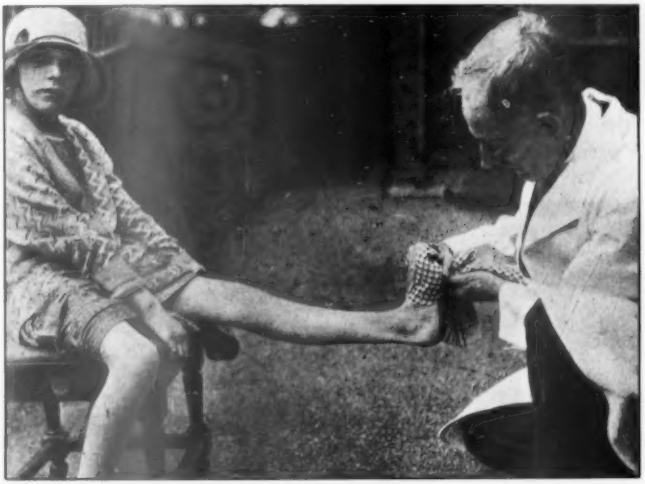
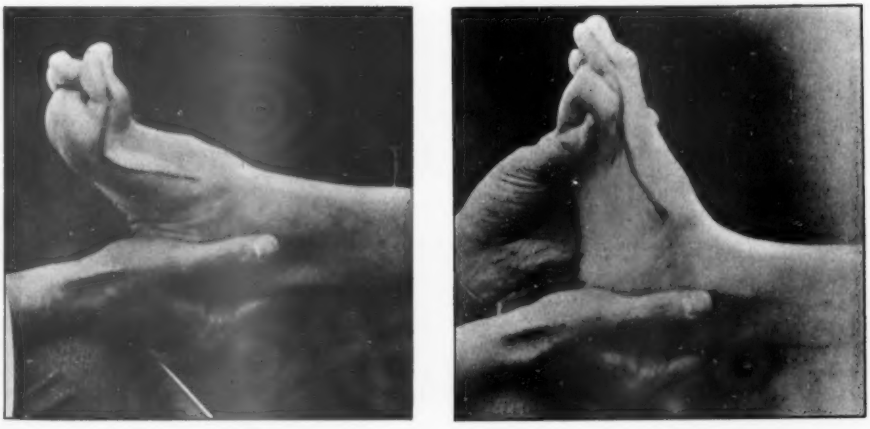


FIG. 2.—Pes cavus, but no contracture of tendo Achillis: it is a fore-foot deformity.



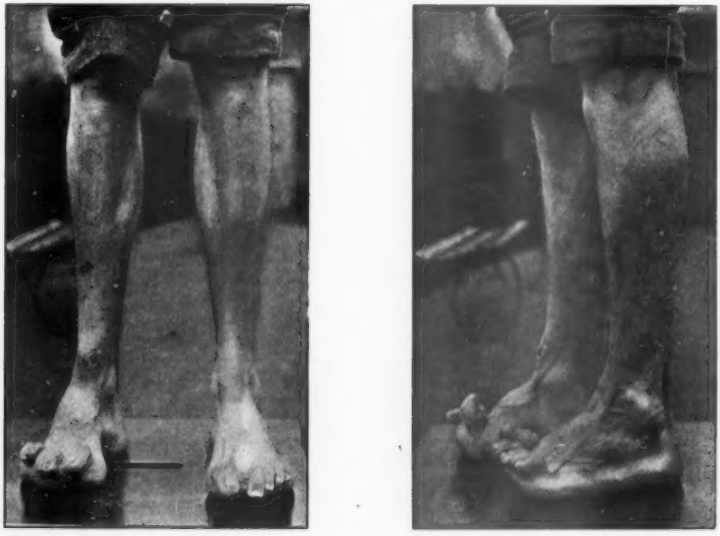
(*a*) (*b*)

FIG. 3.—When the metatarsal heads fall, the toes become retracted and vice-versa; pushing up the anterior arch brings the toes down, and straightens out the metatarso-phalangeal joint.

part played by the interossei is purely passive; the heads of the metatarsal bones drop down below their normal level and so alter the line of action of these short muscles that they necessarily pull up the proximal interphalangeal joints of the toes.

If, in an early case of pes cavus (i.e. one in which secondary contracture of the dorsal parts of the joint-capsules of the toes has not taken place), the anterior arch is pushed well up, the claw-toe deformity wholly disappears; if, conversely, the toes are thrust backward, the anterior arch bulges downward. The two actions are reciprocal. Incidentally, this test illustrates the importance of ensuring, in all these cases, the wearing of adequately long shoes; the shoes must not "fit" the shortened foot; they must allow of the toes stretching forward to their full length when the anterior arch is raised, and it is useless to prescribe an anterior-arch plate, in any case of pes cavus, unless this ample toe-room is provided.

The dropping-down of the fore-foot, then, being a constant and primary feature, even in early cases, would appear to be an important factor in the causation of the deformity; perhaps, even, it is the essential factor. It is remarkable, however, that



(*a*) (*b*)

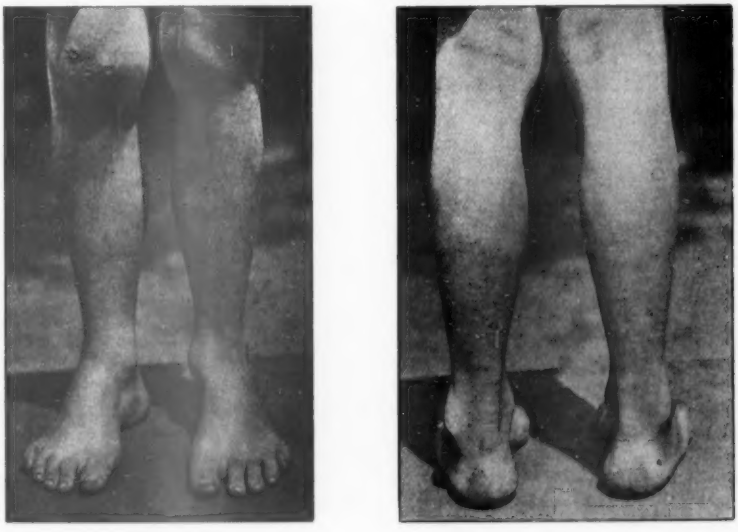
FIG. 4.—A typical case of pes cavus: the patient can pull his toes up, even into extreme hyper-extension, but the pull is not effective upon his metatarsal heads and anterior arch.

pes cavus does not occur with any striking frequency in cases of severe drop-foot due to infantile paralysis. It does occur, but not often. In the majority of cases of pes cavus, there is only a slight weakness of the extensor group of leg-muscles. I first noticed it in a case of transient peripheral neuritis, in a child; the masseuse reported that the extensor muscles gave a normal reaction, on electrical testing, but took an abnormal amount of current; and, on measuring the leg, I found that it was slightly smaller ($\frac{1}{4}$ in.) than its fellow, whilst the extensor muscles were slightly, but definitely, flabby. In this case, unilateral pes cavus gradually developed.

The study of further cases seemed to confirm this first impression. One found that the slight weakness of the extensor muscles sometimes showed itself in an inability to pull the toes up voluntarily as far as they could be pushed passively; in a normal foot, the two ranges are equal. Sometimes, one found that the extensor

communis digitorum was a little weak, whilst the extensor proprius hallucis was normal; in such a case, the outer toes would be somewhat clawed, whilst the big toe would remain perfectly normal (*see* fig. 5). In some cases of pes cavus, the adduction of the fore-foot is a marked feature; these are the cases in which the

FIG. 5.—Clawed outer toes only. In left foot, extensor proprius hallucis is intact, extensor communis digitorum is weak.



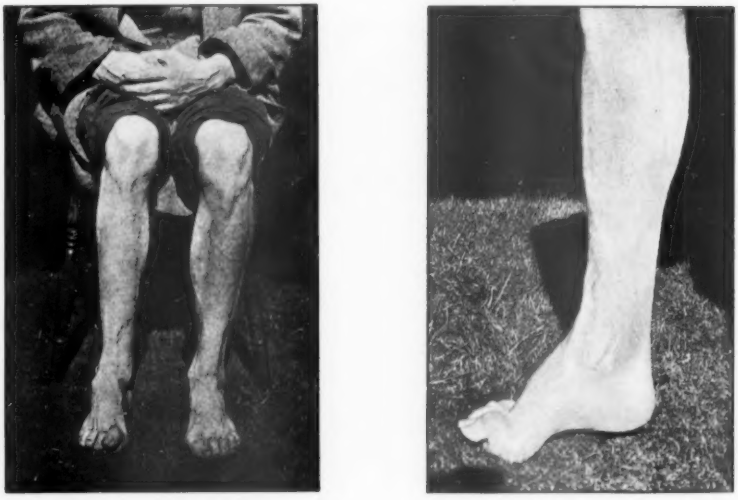
(*a*) (*b*)

FIG. 6.—A case in which the adduction of the fore-foot, and inversion of the heel, are marked.

extensor communis is relatively weak, for this muscle is a strong evertor of the foot as well as an extensor, and when it is weakened, the opposing adductor-invertor muscles pull the fore-foot round (*see* fig. 6). I have never, in many operations, seen

the slightest indication of any contracture, or fibrosis, or indeed of any pathological change, in the short muscles of the inner side of the sole.

Finally, I came across a case which, to my mind, seemed to prove conclusively the causal relationship between weakened extensor action and pes cavus. A mechanic in the Royal Air Force sustained a transverse cut across the front of his right leg, at the junction of the middle and lower thirds (*see* fig. 7). This cut damaged the extensor tendons, but no other structure; some sepsis ensued, and the tendons became more or less bound down, so that their extensor action was much weakened, and the patient was unable voluntarily to pull up his toes, or the fore-foot. A typical right pes cavus developed, with dropped-down fore-foot, plantar callus, retracted toes, and so on. This man was known, of course, to have had normal feet on enlistment, (I have seen his official medical history papers), and the uninjured



(*a*) (*b*)

FIG. 7.

(*a*) Cut extensor tendons, right. No other injury or disease. Result: typical right pes cavus.

(*b*) Side view of same patient. Right foot before treatment.

leg and foot showed no sign at all of pes cavus. This case seems to provide an almost perfect experimental test of the theory that I am advancing.

Murk Jansen, some years ago, suggested that pes cavus should be treated by transplanting the extensor tendons to the necks of the metatarsal bones, and he told me, at the time, that it had given more satisfaction to himself, and to his patients, than all the other methods that he had tried. I tried the method, too, and it seemed to me to do more lasting good than other operations. My idea, in employing the method, was that it afforded a means of relatively strengthening the weak extensor pull; I wanted a direct upward pull on the metatarsal bones, at their anterior ends, and I tried to obtain this by making the extensor tendons pull directly upon the necks of the bones, instead of indirectly, through their attachments to the phalanges.

The ideal operation for the permanent cure of the deformity, in pes cavus, would consist, then, of two distinct and indispensable parts, viz.: (i) the correction of existing deformity, and (ii) the adoption of some measure that would correct the underlying fault in the foot, and prevent recurrence.

The complete procedure is as follows:—

The limb is exsanguinated by a Martin's bandage and an Adams' tourniquet is applied tightly over five layers of lint as high up the thigh as possible.

A curved incision is made on the inner side of the foot, from the front of the heel to the base of the big toe, care being taken to carry it up as high as the tubercle of the navicular; if the incision is not close enough to the bones difficulty is experienced in dissecting under the long arch. The incision is deepened until the upper border of the abductor hallucis is seen; special care must be taken not to undercut the U-shaped skin-flap as this would predispose to delay in healing, from impairment of nutrition. The abductor is dissected away from the bones and joints of the long arch and is completely freed from its origin, behind, from the inner tuberosity of the os calcis and from the internal lateral ligament. In doing this it is important to avoid injury of its blood- and nerve-supply, which enter the muscle quite an inch in front of its posterior attachment. The internal intermuscular septum is excised, the sheaths of the flexor longus digitorum. and flexor longus hallucis (if they are contracted), and all the lesser attachments of the tibialis posticus, the main insertion into the navicular and internal cuneiform bones being left though the tendon is freed right up to the actual bony attachment.

The joints of the inner longitudinal arch of the foot are now freely incised, viz., the astragalo-navicular, the naviculo-cuneiform, and the cuneo-metatarsal. The knife must enter the joint freely on the upper, internal and lower aspect of each, the tendons of the tibialis anticus and posticus being lifted up for the purpose. The astragalo-navicular ligament should be cut last; the surgeon should obtain the main correction at the anterior joints and only incise the astragalo-navicular ligament as far as may be necessary; indeed, in mild cases it may not be necessary to cut this structure at all.

All this allows of a gaping open of the joints on the inner side of the foot, and a relative lengthening of its inner border in proportion to the outer. The Steindler method alone does not provide this, but it is necessary if a perfect correction of the deformity is to be attained.

The knee is now fully flexed and the foot placed on its inner surface, on a sandbag. The ordinary external Steindler heel-cut is made, and all the structures of the sole which are attached to the os calcis are freed from it. The short deep fibres of the lesser calcaneo-cuboid ligament are also thoroughly divided.

Alternatively the original curved mesial incision can be prolonged upward and the Steindler operation can be completed through that. But the original external incision appears to me to be easier, and to give a more thorough dissection of the structures of the sole.

The effect of the Steindler incision is to allow all the structures of the sole to be lengthened and the foot is now forcibly twisted upward and outward into an exaggerated valgus position so as to break down any half-divided structures.

This open operation is immeasurably superior to any of the older closed methods, in that it allows of the greatest possible correction of deformity, in every structure that it is permissible to divide, with a minimum of damage to the remainder. The inner longitudinal muscles of the sole are lengthened, for example, without impairment of function, and the plantar fascia can be elongated by three-quarters of an inch, or more, without the infliction of those tender lumps in the sole that so often follow plantar fasciotomy.

This ends the first stage of the operation, i.e. the correction of the existing deformity. Nothing has been done as yet to remedy its cause, or to prevent relapse.

That is the object of the second stage.

The operator, sitting opposite the sole, now makes three incisions along the first, third and fifth metatarsal shafts. These incisions are deepened directly to the bone with the least possible amount of lateral undercutting of the tissues. The tendons of the

extensor communis digitorum are cut, as far forward as possible. The necks of the first, third and fifth metatarsals are clearly exposed by rugining the soft parts off them; a hole is bored transversely through each and it is carefully enlarged and smoothed with a reamer. A salmon-gut stitch is passed through the extreme tip of each tendon; an awl, with a hole at the end of it, is passed through the metatarsal bone, and the tendon is drawn through, by threading the salmon-gut suture through the eye of the awl and then withdrawing the latter instrument. To save trouble, and to give the greatest possible lift to the centre of the anterior arch, two tendons are threaded through the third metatarsal, and the others through the first and fifth respectively. (The fourth metatarsal is usually very delicate in structure and rather difficult to reach.) Each tendon is held at the tip by an Ochsner forceps, to prevent its retracting, as it is threaded through the bone. When all have been threaded the permanent suturing is begun. Throughout this process the foot is pushed as strongly as possible by an assistant in an upward and outward direction; each tendon-end is twisted once, or even twice, round the proximal part of the tendon; it is then pulled as taut as possible and is sewn, through and through, with fine salmon-gut. (This material is preferred because it more closely resembles normal tendon than any other; it is very strong and lasting, and never causes the slightest reaction in the tissues if it is put in by the no-touch technique.) The twisting of the tendon round itself greatly decreases the risk of the suture-material pulling through under the strain.

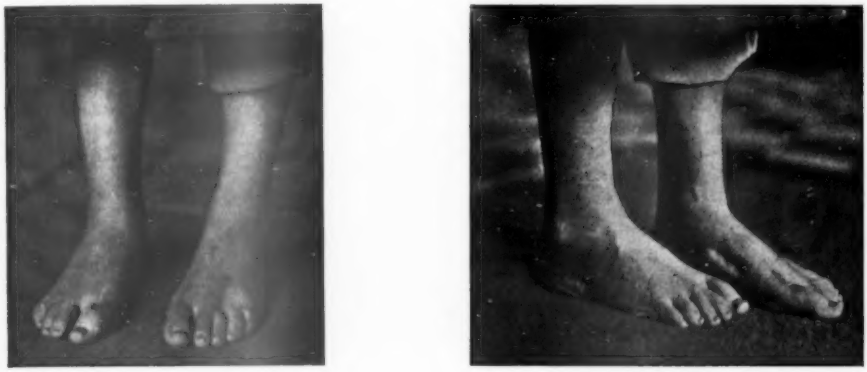
The three incisions on the dorsum of the foot are now closed with a continuous stitch of gossamer salmon-gut, on a No. 20 half-circle needle. A zig-zag stitch must not be used, in spite of the extreme thinness of the skin here, because it puts the skin under so much tension as to involve a risk of sloughing. A continuous stitch, however, which just brings the edges into apposition, is perfectly satisfactory. The outer heel-cut can be closed with a medium-sized suture, and presents no difficulty. The curved mesial incision is under tension now that the inner border of the foot has been considerably lengthened, and it is therefore essential that skin-stitches should be put in, close together; the greater the number of sutures, the less is the strain upon each, and the greater is the likelihood of their holding.

If the pulling-up of the metatarsal heads has brought the toes down into a satisfactory position, this ends the operation. But if they do not come down properly, owing to contractures from years of malposition, it is well to ankylose the proximal interphalangeal joints in full extension. The little toes are often drawn right up, on to the dorsum of the foot, and are quite functionless; very often they cannot be brought down or rendered functional; in that case it is best to amputate them.

A double thickness of sterile Gamgee tissue is wrapped closely round the foot, and is bandaged on with a firm flannel bandage. Then (and only then) the tourniquet is removed. I prefer not to use any splint; if the tendon-suturing has been efficient, it will withstand the strain. If there is any oozing afterwards, from the mesial wound, it is dried by copious applications of 5% salicylic acid powder. As soon as the wounds are strongly healed the patient is encouraged to walk, so that the weight of the body may stretch the recently divided structures and maintain the correction.

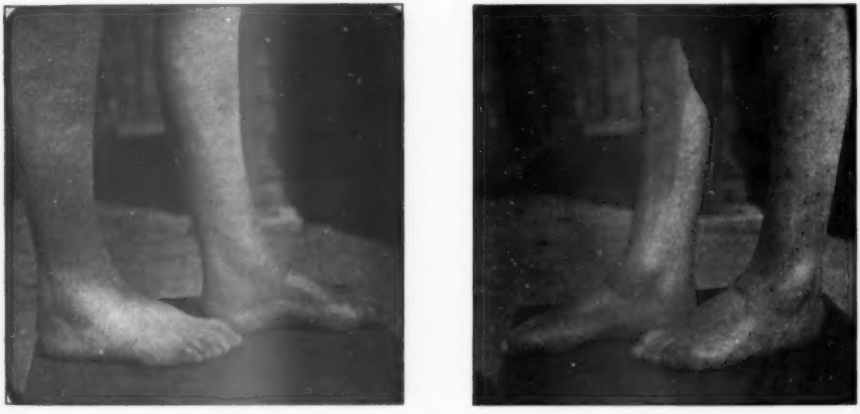
I have not yet quite decided the best age for performing this operation. It is certainly appropriate in adults and adolescents, and my practice, hitherto, has been to temporise, with milder measures, until the patient has attained the age of 16 or 18 years, and then carry out this final operation. In patients who are under regular supervision, the most important treatment in the interval is the routine stretching of the toes. It has been suggested to me that in neglected cases in younger patients, when the toes have already become much retracted, the "final" operation should be performed earlier. I am testing this suggestion at the present time. The appearance of thick horny plantar calluses is certainly an indication for the performance of the major operation without further delay, at any age after about 12 to 14 years; for, if treatment is postponed the calluses become so septic that they gravely endanger the extensive operative procedure described. (Incidentally, if such thick, painful plaques of callus are already present when the patient is first seen, the best treatment, I think, is to cut them away to a large extent, and then give fairly big doses of X-rays.)

Children seem to vary very much in the extent to which they complain of pes cavus. But in adults it is always a seriously crippling condition, and active treatment should be given to every patient who complains of pain. The surgeon to the City Police told me, some few years ago, that he had found that pes cavus was a far more disabling condition than flat foot; if a constable nearing the retiring age began to complain of weak feet, it was usually possible, by giving him suitable



(*a*) (*b*)

FIG. 8.



(*a*) (*b*)

FIG. 9.

Two cases illustrating result of the complete operation, as described.

duties, to " nurse " him through the remaining years of service; but the slightest sign of pes cavus invariably necessitated the immediate invaliding of the sufferer at any age.

The operation that I have described is appropriate to adolescents and to all adult cases but the septic and the ultra-contracted and fibrotic. Special care must

be taken, of course, in cases of pes cavus due to infantile paralysis; it is useless to transplant extensor tendons, if their muscle-bellies are paralysed.

There remains only the question of the relief of elderly persons with very contracted feet of what I may describe as the "shrivelled" type. These persons are unsuitable for the complete operation, but they press for something to be done for them. A vigorous stretching under anæsthesia—perhaps even the cautious application of the Thomas' wrench—seems to afford satisfactory relief.

> In one striking case, a warder in a gaol was threatened with compulsory retirement, owing to disability from bilateral pes cavus, which he had had for years. He could scarcely hobble round the corridors, and it was difficult for the doctor to suggest even light duties that he could perform efficiently. The man clamoured for "something to be done," so, more to satisfy him than for any other reason, a thorough wrenching was carried out. No material improvement in the appearance of the feet resulted, but the patient said that his feet were now much more comfortable, and he performed full duty for several years afterwards, before retiring at the ordinary age.

This result has been confirmed in one or two similar cases, and the method seems to be worthy of a trial, in advanced cases in elderly persons.

The results of the ordinary standard operation that I have described, above, seem to be quite permanent. The toes, if not quite down after operation, tend gradually to come down, and to regain their function; pain under the anterior arch, and callus, disappear, and the walking-capacity improves greatly. Patients have reported that they are able to walk "for miles," and they are found to be able to follow arduous occupations, such as that of farm-labourer. The theories upon which the treatment is based may or may not be correct; but theory matters little, provided that the practical outcome stands up to the test of use; and I submit that this operation provides a rational, effective, and permanent cure for pes cavus. No claim is made to originality, in any stage of the operation; all its component parts have been used before; what I have done—if anything—is to assign each to its proper place and function, and to take a heap of heterogeneous materials, and build with them a solid, lasting, and scientifically designed structure.

Section of Medicine

President—Sir FARQUHAR BUZZARD, Bart., K.C.V.O., M.D.

[*October* 23, 1934]

DISCUSSION ON THE ULTIMATE PROGNOSIS OF CORONARY OCCLUSION

Professor John Hay: The ultimate prognosis in coronary occlusion means, I take it, the prognosis in those patients who survive the initial attack—the remote, rather than the immediate, prognosis—and it must deal both with the expectation of life and the expectation as to efficiency.

Prognosis—forecasting the future—is always difficult, and that of coronary occlusion is recognized as especially hazardous.

There is, of course, the statistical approach, and this affords information of value, but its usefulness is primarily actuarial. Statistical findings are, nevertheless, serviceable in correcting general impressions—impressions concerning the importance or otherwise of certain symptoms which may, by reason of their dramatic character, have unduly impressed the clinician.

The survey of a long series of cases and a careful scrutiny of the published records do, in the long run, help us to assess with some confidence the true prognostic value of the various factors on which prognosis is based.

Valuable as statistics are, however, I imagine we are all agreed that there are two essentials in arriving at an opinion as to the future of a patient who is suffering from coronary occlusion. First, we must have a clear conception of the actual conditions in the heart, the underlying pathology, the changes which precede and follow on the coronary block. And, secondly, we must be familiar with the life-history of this particular ailment, this particular cardiac accident. We must be able to visualize the clinical picture in all its phases and possible developments. Then and then only can we attempt, with reasonable chance of success, to assess the significance of the signs and symptoms as they arise and translate them into terms of prognosis.

First, then, the fundamental pathology. Probably a widespread arteriosclerosis, blocking of a branch of a coronary artery, with ischæmia and death of the myocardium involved. The necrosed myocardium may rupture, but usually slow cicatrization occurs and by the end of two months the necrotic myocardium is replaced by scar tissue. The future efficiency of the heart depends upon the extent of the necrotic area and the relation of this area to the rest of the myocardium, and also on the integrity of the remaining myocardium.

The clinical picture is familiar, but certain features can with advantage be stressed, since they have a bearing on prognosis. Coronary occlusion may come suddenly and without warning, or preceding the occlusion there may have been an increasing liability to anginal pain, or cardiac failure of the congestive type. It is characteristic that the attack is spontaneous although it may be precipitated by physical effort. The patient may die suddenly from shock or ventricular fibrillation at the onset. Death may occur within a few weeks from rupture of the heart. Embolism, cerebral or pulmonary, may cause death. Cardiac failure of the congestive type may develop rapidly subsequent to the occurrence of the occlusion and progress relentlessly to a fatal issue.

Those who survive the shock of onset and the earlier complications do so with a gravely damaged heart, and those factors which predisposed to the original occlusion still persist and are as potent as before. On the other hand, the patient has had his lesson, he is probably under medical supervision, and is prepared to play the game and live within his limitations.

The chances of surviving the first attack are difficult to assess because many of the milder cases of coronary occlusion escape diagnosis and those who die suddenly at the onset of an attack are frequently not included in the recorded figures.

The *initial* mortality is, without doubt, heavy. Carey Coombs [1], in a total of 144 patients, reports 49 deaths in or shortly after the initial attack—equalling 34% or one-third of the total: and he further records that of the surviving patients 32 died within the subsequent year—which makes a total of 81 deaths within the twelve months. Conner [2, 3] gives the mortality during the first attack as low as 16·2% (46 deaths in 287 patients). On the other hand, S. A. Levine [4] takes a gloomier view of the immediate prospects. He finds that only 50% have an immediate recovery. Parkinson and Bedford [5] in 100 cases (which, however, do not include those patients who died suddenly at the onset) found that 23 died during the first six months.

I do not think that anything is to be gained by quoting further figures in regard to the onset, and in any case we are now more concerned with the fate of those who survive the perils of the first attack and its immediate results.

Those who survive are sorely crippled and it is only too true that sooner rather than later they will die a cardiac death, either from congestive failure, angina pectoris, or a further coronary thrombosis.

For the purposes of discussion the survivors can be arranged in two groups:—

Group I.—Those in whom the cardiac disability is shown by an increasing tendency to dyspnœa and a gradual drift towards failure of the congestive type. Pain is *not* the dominant feature in these patients: but there is orthopnœa, Cheyne-Stokes respiration and, subsequently, pulmonary œdema, anasarca, ascites, hydrothorax, complete the picture. In this group should be included those whose cardiac reserve is still further hampered by the presence of auricular fibrillation which may have originated with the coronary occlusion or have developed later.

The outlook for all patients in this group is bad from the start. It is true that something may be done with digitalis for the fibrillators, but for the other patients treatment is chiefly symptomatic and the outlook is ominous. Their future is measured in *months*.

Group II.—Those who make a satisfactory recovery and are able to lead reasonably useful lives. They are rarely entirely free from some form of cardiac distress and this usually takes the form of anginal pain rather than of dyspnœa. They are pulled up by angina of effort and must learn to live within their limitations. It is the patients in this group who surprise us by their longevity. When they die it is true that they succumb to some type of cardiac defeat—possibly a further coronary occlusion which comes on spontaneously and is perhaps as unexpected as the first

attack, or they may die in an attack of angina pectoris. A relatively small percentage of these cases drift into a condition of congestive failure, and there is always the remote chance that some intercurrent disease or vascular accident may prove fatal. In this group the future is measured in *years*.

There is one point, at any rate, concerning which we are all in agreement. Our clinical experience of the last five to ten years justifies a far larger measure of optimism than we formerly dared to express.

CONNER AND HOLT. JULY 1930.

287 Cases: 117 Alive.

Alive after:		During:	Death
1 year ...	88	2nd year ...	23
2 years ...	65	3rd ,, ...	16
3 ,, ...	49	4th ,, ...	9
4 ,, ...	40	5th ,, ...	15
5 ,, ...	25	6th ,, ...	8
6 ,, ...	17	7th ,, ...	2
7 ,, ...	15	8th ,, ...	8
8 ,, ...	7	9th ,, ...	3
9 ,, ...	4	10th ,, ...	0
10 ,, ...	3	11th ,, ...	1
11 ,, ...	2	12th ,, ...	1
12 ,, ...	2	13th ,, ...	1
17 ,, ...	1	18th ,, ...	1

These statistics give an idea of the outlook and include some interesting examples of patients who survived the initial attack by many years. Connor and Holt found that in more than half of their cases (67%) there was only one attack of occlusion: if a second attack did occur then it did so within the first twelve months. White and Bland [6] in an account of 200 cases of coronary thrombosis report that 33 died in less than one year after the initial coronary occlusion, and 33 survived the first attack by four years or more—that is, 16½%. Levine [4] in his monograph states that in his opinion the average expectation of life of those who recover will be about three years. In his series of 145 cases—11 lived for more than three years after the first attack; of these, seven were alive at the time of publication, six lived for four years or more, and two were alive more than five years after the attack. In Parkinson's and Bedford's figures, of the 100 cases (not including those who died suddenly at the onset of the coronary thrombosis), eight were leading an active life; 33 a comfortable, though restricted, life; 25 completely invalided owing to pain and failure.

If the first stormy and critical stage can be weathered then there is an expectation of from two to five, or even more, years, and there are some remarkable cases on record which far outrun these figures.

In John Hunter we have the classical example of a useful life lived at high pressure in spite of coronary occlusion. His first attack was both severe and alarming. It occurred in 1773, when he was 45 years of age. Three years later there was a second attack, and during the last seven or eight years of his life he was liable to recurrent attacks of effort angina, in spite of which he carried on his strenuous life—"less fit for work, yet ever engaged in it." In 1793 he died suddenly, having survived his first attack of coronary thrombosis by twenty years.

As other notable examples of longevity after thrombosis, two cases are reported by Paul White [7], and deserve especial mention:—

(1) The first is that of a man who was alive in March 1932, aged 67, twenty years after his first attack of coronary occlusion, having survived two further attacks. He was a minister and at the time of the last note was alive and active, preaching every Sunday, although suffering from some substernal oppression or paroxysmal dyspnœa.

(2) White's other case was that of a man who lived seventeen and a half years after an attack of coronary occlusion and finally died at the age of 80 from apoplexy without cardiac failure. At the age of 72 he climbed mountains with ease and even at the age of 80, shortly before his death, he could walk five miles without discomfort. At the autopsy there was found to be a "firm scar in the heart muscle resulting from the healing of the infarct." The heart was otherwise in good condition.

The figures given by Conner (*see* table), were interesting and most cheering, particularly in regard to the relatively large number of patients who were alive at the end of the fifth year: (namely 21% of the 117 survivors), and at the end of 10 years, 3·4%.

Among my own records there are a number of patients who have pleasantly astonished me by living longer than at first seemed probable.

C., died in February 1934, thirteen and three-quarter years after a severe attack of coronary thrombosis which occurred when he was aged 70. He retired from active practice, at my advice, and lived a happy and useful life—although liable to slight angina of effort on over-exertion.

January 1933: A second thrombosis, from which he recovered.

February 1934: A third and final attack.

B. This patient suffered from aortic obstruction and slight regurgitation.

First attack, December 1913, when aged 54.

Recovered and lived the life of a Consul in various parts of the world. 1915: a second attack. 1924: a third attack.

He died in January 1925, more than eleven years after the first attack: he was always liable to some anginal pain on effort or excitement.

H. S. First attack in 1922, at the age of 57.

Attack lasted for several days.

Second attack, 1928.

From these he recovered and led an ordinary life as an accountant. Then about nine months before his death he began to suffer from effort angina.

He died suddenly, nine and a half years after the initial seizure.

A. C. First attack (severe) July 1926 at the age of 58.

A further attack in May 1930.

He recovered slowly but for the last three years has been carrying on his practice, with assistance.

G. P. When aged 63 a typical attack in October 1928.

Recovered and able to lead a quiet life. He had retired from active practice in 1925. Some substernal pain on effort.

May 1934, a second attack from which he has recovered.

A. H. First attack when aged 49, in December 1928.

He is alive, almost six years later, and fairly comfortable.

N. S. At the age of 66 a typical attack, December 1928.

For the last two years he has been practising as a doctor, but taking life more quietly because an undue effort induces anginal pain.

He has not had a second attack.

On careful scrutiny of my case records and those published by others, two facts stand out: (1) The frequency with which life is ended suddenly—presumably by a terminal (coronary) thrombosis; (2) the ominous significance of a progressive tendency to dyspnœa and congestive failure.

Unfortunately there do not appear to be any signs or symptoms which surely indicate an impending occlusion, unless it be an increasing liability to effort angina. We know that those factors which in the first instance predisposed to coronary occlusion are still present and that the liability to occlusion has been unmasked by the very fact of the initial attack. We know also that the liability to another attack diminishes with time for, as Conner showed, 50% of all second attacks occur within twelve months of the first. The onset of the second is, however, as sudden, as dramatic, and as unexpected as in the original attack.

We are on surer ground in regard to the prognosis when there are indications of cardiac failure. The outlook is indeed grave when unequivocal signs develop in a patient who has otherwise made a satisfactory recovery from the initial occlusion and whose cardiac condition has become stabilized. Weakening of the heart sounds, a gradually increasing tendency to dyspnœa, orthopnœa, Cheyne-Stokes breathing, and signs of congestive failure, are sure indications of grave damage. The onset of auricular fibrillation may in itself be sufficient to precipitate failure and then the immediate prognosis depends on the response to digitalis. More often failure develops gradually with a normal fundamental rhythm. It is then the " writing on the wall " and must be accepted as ominous. A few months, possibly a year, is all that one can hope for, even with every therapeutic aid.

What other factors or conditions may modify the prognosis?

Age at onset.—Levine's figures indicate that the younger are more apt to recover from the thrombosis and, having weathered the attack to live longer.

Blood-pressure.—I cannot satisfy myself that any reliable inference can be drawn from the blood-pressure findings.

If anything, those with the lower pressures fared worse than those with pressures on the higher side. Nevertheless, it is reassuring to watch a pressure approach the normal from the hypopiesis following a coronary occlusion.

A diminishing pulse pressure, with other signs of cardiac failure, is obviously a serious finding.

Electrocardiograms.—These are of more value in the diagnosis of coronary occlusion than as aids in prognosis.

Low voltage—unsatisfactory in itself, is not uncommon in those who have survived a coronary thrombosis by several years.

Branch bundle block and complete block are naturally of adverse significance, since the localized lesion is usually associated with a more diffuse fibrosis.

Carey Coombs, and White and Bland, are agreed that the presence of angina of effort prior to the coronary occlusion makes little or no difference to the outlook. This is worth further investigation.

There is another point on which I should welcome the opinion of members—namely the significance of glycosuria. In Levine's series of 145 patients, 34 were glycosurics. This complication or coincident condition did not modify in any degree either the age at which coronary thrombosis occurred, or the prognosis. In my own experience the glycosuria has usually been of a mild type and has not appeared to add to the difficulties or diminish the expectation of life.

Finally, the outlook will depend largely on the extent to which the patient is prepared to co-operate with his doctor. No patient can afford to treat his damaged heart with disrespect, and experience has taught us that a damaged heart will often do its duty for years when treated with the kindly consideration which is its due.

It is of interest to remember that ten years ago a discussion on the ultimate prognosis of coronary occlusion would have been futile. We had not then the

necessary data on which to base an opinion. In the last decade we have learnt much and it is fitting that we should now pool the experience which we have so recently gained.

REFERENCES

1 COOMBS, CAREY F. "Prognosis in coronary thrombosis." *Bristol Med.-Chir. Journ.*, 1932, xlix, 276.
2 CONNER, LEWIS, A. (New York). "Prognosis in coronary thrombosis." *Lancet*, 1930 (ii), 83.
3 CONNER, L. A. and HOLT, EVELYN. "Subsequent course and prognosis in coronary thrombosis." *Amer. Heart Journ.*, 1930, (v), 705.
4 LEVINE, S. A. "Coronary thrombosis: its various clinical features." Baltimore, 1931.
5 PARKINSON, JOHN, and BEDFORD, D. EVAN. "Cardiac infarction and coronary thrombosis." *Lancet*, 1928 (i), 4.
6 WHITE, P. D., and BLAND, E. F. "A further report on the prognosis of angina pectoris and of coronary thrombosis." *Amer. Heart Journ.*, 1931, vii, 1 (October).
7 WHITE, PAUL D. "Longevity after coronary thrombosis." *Journ. Amer. Med. Assoc.*, 1933, 233.

Mr. T. F. Cotton: The coronary occlusion syndrome is not difficult to recognize when the patient is seen during the acute illness. The clinical features are characteristic, the diagnosis is not often in doubt, and the after-histories of those who die in the attack are easily obtained. It is said that death occurs rapidly in half these cases during the acute illness. To accept the statement that 50% of patients die at the time of acute coronary obstruction, I think we must include among them some who die from a recurrence of coronary thrombosis. We are on less certain ground when we attempt to forecast the course of the disease in those who recover from acute coronary obstruction, for after-history records—both of private and hospital patients—extending over a period of several years, are usually incomplete.

A good deal of useful information regarding the course of this malady may be obtained from the past history of patients who present themselves for examination because of anginal pain. The coronary occlusion history is distinctive and we should be able to recognize with a fair degree of accuracy a past history of coronary occlusion. Severe anginal pain, lasting for several hours, unrelieved by nitrites, supported perhaps by other symptoms of occlusion—dyspnœa, vomiting, pyrexia, low blood-pressure in a hypertension subject—is good evidence. Residual changes in the electrocardiogram are confirmatory proof of past occlusion. It is well known that in a patient who has had coronary occlusion an angina of effort may develop.

What I have to say concerning the ultimate prognosis in coronary thrombosis is based on a survey of 200 patients with anginal pain whom I have seen in private practice. In 73—more than one-third of these—there was a history of coronary occlusion in the past, or they had acute coronary obstruction at the time of examination. In 33 cases, or 45%, I noted that anginal pain followed coronary occlusion. Increasing disability from angina of effort, spasmodic angina or recurring thrombosis seems to have been the fate of many of these patients. Twenty out of the total number of 73 I know are dead. There were eight with auricular fibrillation, paroxysmal tachycardia, and heart-block, as a direct result of the occlusion; five of these died within a fortnight of the attack, and three recovered from the acute illness. Death was due to a recurrence of coronary thrombosis in five cases.

Of the 40 patients who did not develop angina as a result of the occlusion, 16—or 40%—made a good recovery; one is alive and well after nine years.

The after-histories in this series of cases are incomplete, and post-mortem evidence is lacking; one must be guarded in drawing conclusions from the figures I have given. They have some value I think in determining what happens to the

patient with coronary thrombosis. I believe I am justified in saying that a large number of patients who survive the acute illness become anginal subjects and die anginal deaths ; and those who survive the accident of acute obstruction and do not develop angina, will probably make a good functional recovery.

I have no concrete evidence that the prognosis is graver in angina following occlusion, than in angina in other cases, apart from the syphilitic group. The fact that I have never observed a good functional recovery in angina after occlusion, as I often have in the others, leads me to believe that the prognosis is graver in the former than in the latter.

Dr. B. T. Parsons-Smith : It is encouraging to contemplate the title of this discussion in that it implies the possibility of recovery from a catastrophe which, until moderately recent times, was regarded as invariably fatal.

As the result of accurate clinical investigation on the part of many workers we now possess a reasonably clear-cut conception of coronary thrombosis, and, though the differential diagnosis of the condition is at times a matter of difficulty, one cannot fail to be struck by the universally accepted prevalence of the malady, even allowing for the fact that errors may occur, the disease being still in its relatively early and fashionable stages.

In order to frame anything approaching a definite opinion in regard to the ultimate outlook of those patients who survive the immediate effects of an acute coronary occlusion, we must be prepared to balance not only considerations of a general type but also any such specific information as may be available of the progress of the disease. The general considerations are those which apply automatically to all cases of cardiovascular disability and include the questions of heredity, age, temperament, nutrition, habits, mode of life, etc. In conjunction with these we should most carefully investigate all the clinical evidence obtainable of the progress of the acute attack, more particularly with a view to forming an opinion of the approximate size of the myocardial infarct, in regard to which the principles of prognosis must obviously involve a speculative assessment. Further, we need to consider the general scheme of progress during all the phases of the acute illness and the convalescent period, the possible occurrence of some one or other of the well-known complications which characterize the disease, and the patient's response to such treatment as may have been required. Equally important with the above is an estimate, as accurate as possible, of the myocardial efficiency and circulatory reserve which may be available after recovery from the early stages of the illness.

In this latter respect it would be impossible to overestimate the assistance which electrocardiograms afford ; curves in the more favourable cases provide convincing proof of the transient nature of the myocardial incapacity following coronary thrombosis and, conversely, serial records demonstrating permanent discrepancies may be equally illuminating.

We should moreover pay particular attention to the subjective symptomatology, which may vary from a trifling degree of effort syndrome to serious manifestations of circulatory embarrassment—breathlessness, giddiness, nocturnal dyspnœa, physical fatigue, pain, etc. The development of these, or their degree of aggravation following an attack of coronary thrombosis, will afford valuable proof of the progress of the disability and its ultimate outlook ; in a large proportion of cases retrosternal pain will be complained of as a prominent symptom, it may precede the actual attack of thrombosis and its later occurrence must always be viewed with alarm, for, in direct proportion to its severity, it is an indication of myocardial ischæmia and probably progressive dystrophy.

Complementary to an adequate assessment of the subjective symptomatology a comprehensive study of the physical signs is essential to a correct understanding of the course of the disease and the complex problem of its ultimate prognosis. Unfortunately we have no reliable method of estimating the functional capacity of the heart in anything approaching concrete terms, apart from which we must always be prepared to realize that, following such an event as a coronary occlusion, numerous variables and unknown factors may arise to complicate the issue and obscure the prognosis. For these reasons, and even admitting that valuable statistics are available nowadays in regard to the ultimate outlook of coronary thrombosis cases, we shall be well advised to investigate each case on its individual merits, noting such discrepancies as may be obvious and as far as possible estimating their significance. The well-known signs of circulatory incompetence may proclaim their presence in unmistakable form and with varying grades of severity. Certain of these are definitely indicative of an unfavourable outlook—enlargement of the heart, persistent enfeeblement of the heart-sounds, tachycardia at rest, paroxysmal tachycardia, a subnormal arterial and a raised venous pressure, a diminished range of pulse pressure and pulsus alternans. A further manifestation, and one, moreover, which invariably signifies grave myocardial incompetence, is a diminished cardiac excursion which may be suspected during the clinical examination and confirmed by inspection on the X-ray screen. We should inquire for the presence of this sign in all cases, remembering that some degree of myocardial inertia is a probable development following coronary thrombosis and that its persistence may be interpreted as of serious moment, more particularly if it happens to feature an otherwise well-defined failure syndrome.

A further development which frequently characterizes the after-history of thrombosis cases is cardiac arrhythmia, and to this we must more often than not devote special consideration. All the known varieties of arrhythmia and embarrassments of intracardiac conduction have been noted following coronary thrombosis and, as a general rule, their significance, except in the initial stages of the illness, may be assessed on the well-accepted basis to which these disabilities conform in cardiovascular disease. In a certain proportion of cases the infarct extends to the interventricular septum, thereby inducing heart-block, partial or complete, and this condition may materially influence the course of the disease; its early development need not occasion alarm; in fact we have reason to believe that the bradycardia of heart-block may be, up to a point, a valuable and conservative process in that it counteracts the tendency to ventricular tachycardia, which is so frequently a disquieting feature during the acute phases of the disease and its subsequent convalescence. In this context one should emphasize the more favourable outlook of young patients in whom the idio-ventricular rate of heart-block is moderately high (? 50-60), as opposed to the elderly patients in whom the ventricular rate is fixed at a definitely lower level (? 28-40).

We may summarize as follows:—

In 50 to 60% of all cases coronary thrombosis is immediately fatal, and a relatively small number of patients (? 5 to 10%) recover completely; the remainder (roughly 40% of all cases) may be expected to make a partial recovery, and their prognosis may be determined to some extent by careful investigation of the circulatory efficiency and its reserve capacity, always remembering that complications may develop in a disease of which the pathological basis is coronary atheroma associated with myocardial dystrophy.

Sir Maurice Cassidy: Professor Hay has said that after a coronary occlusion those factors persisted which had brought about the thrombosis, and that further clottings were inevitable sooner or later. I submit that there is at least one factor

in the ætiology of a coronary occlusion which is sometimes amenable to treatment, namely, that of infection. Surely in many thromboses, either in the coronary circulation or elsewhere, there is an infective factor as well as an atheromatous or arteriosclerotic one. The pyrexia and leucocytosis which so often accompany a coronary thrombosis are evidence of such a factor, and if the primary infective focus can be dealt with, subsequent thromboses are less likely to occur. In a young-middle-aged subject, for example, when there is definite dental infection, extractions cautiously carried out after all acute symptoms have subsided, may favourably influence subsequent progress.

I was glad to hear that the openers of this discussion did not take electro-cardiographic changes too seriously when making their prognosis. We have all seen patients who have been almost symptomless for years after a coronary occlusion, in spite of the presence of gross electrocardiographic changes. Even in the presence of a complete auriculo-ventricular heart-block following a coronary occlusion a surprising immunity from symptoms may occur.

Dr. D. Evan Bedford : Some information from the point of view of prognosis is to be obtained from the pathological findings. Coronary thrombosis is seen most frequently in post-mortem specimens at two stages; first, in the early stage, before an infarction had had time to form, and secondly, in the chronic stage of fibrous infarction. In other words, patients with coronary thrombosis frequently die at the onset, or recover from the acute attack and die later from some form of cardiac failure. We are not concerned in practice with patients who die suddenly at the onset, but only with patients in whom coronary thrombosis is diagnosed during life. From the figures cited by Professor Hay, and from personal statistics, I estimate the mortality during the acute attack of coronary thrombosis as not more than 25%, that is to say, a patient seen alive with coronary thrombosis has a three-to-one chance in favour of recovery. With regard to the remote prognosis, sufficient information is not yet available, as in all reported series of cases a large proportion of the patients are still living; when all these patients still living have died, their duration of life will prove to be longer than present figures indicate.

It is of some importance to know exactly what should be said to the relatives in a case of coronary thrombosis. In every case life is in danger and no single sign or symptom is of much help in deciding the outcome in the early stages. One must be satisfied to say that life is in danger, but that the chances of recovery are good. Once the twentieth day is past, the likelihood of serious complications arising greatly diminishes, and the patient's chances are definitely better once this danger period is over. It was not always the patients whose recovery was most complete who lived longest. A man, aged 60, had a calcified infarct of the heart, diagnosed in life by means of X-rays, and confirmed by post-mortem examination. This patient was under observation for five years with low blood-pressure and incessant attacks of angina, and later dyspnœa. Generally speaking, a blood-pressure that persisted in the region of 100 mm. systolic was of serious omen for the future.

Mr. T. Skene Keith said he would like to approach the subject from the other end, that is, knowing the diagnosis and, incidentally, the prognosis, to look back along the history.

In a recent series of 200 autopsies, carried out in public mortuaries at the request of the coroner, there were 110 cardiovascular deaths, and of these 51 were due to coronary disease.

Of these 51, 24 showed the presence of definite clot, while 14 had old fibrous infarcts. Of these 14 with infarcts, eight also had recent clots. From the point of view of prognosis this suggested that 14 cases out of 51 had had a definite previous attack.

As to the history: 21 cases provided no more than that they had either collapsed suddenly and died, or that they had been found dead in their rooms. In nine cases there was evidence that up to the time of death they had been in reasonable health, and in 6 that there had only been a vague story of indefinite ill-health.

Of the remaining 15 cases, nine had suffered from pains in the chest, four from indigestion, and two from heart disease unspecified. However less than a third of even these cases had consulted a doctor.

Of these 51 cases, therefore, 14 showed signs of a definite previous attack, and 15 had suffered from symptoms which might or might not have been anginal in nature.

He (Mr. Keith) further called attention to the value of a determination of the viscosity of the blood in prognosis. It appeared, from a limited number of cases examined, that a high viscosity predisposed to anginal pain which was relieved by reducing the viscosity. It was probable, therefore, that patients with an increased viscosity would have a worse prognosis than those with a more fluid blood.

Dr. Philip Ellman said that he would be grateful if Professor Hay would elucidate certain problems which had occurred to him in connexion with the prognosis of coronary thrombosis.

The first was with regard to electrocardiography, especially in connexion with the prominent Q waves in lead III, sometimes transient and sometimes persistent. He had recently seen a case of acute coronary thrombosis in which there had been a typical deviation of the R T period in lead III where both Q2 and Q3 waves had persisted, even when the deviation or high take-off of the R T segment had returned to normal. This had, in fact, persisted for a year after the acute attack, the other electrocardiographic changes having returned to normal. He wondered whether the persistence of Q2 and Q3 (the persistence of Q3 alone was, of course, not uncommon) for some time after an acute attack, when the other abnormal changes had been rectified, was of any value in prognosis.

A second problem was concerned with the relative prognosis of coronary thrombosis occurring in: (*a*) a comparatively healthy heart and (*b*) a diseased heart. In the case of angina pectoris it had been suggested by some observers that the prognosis of angina of effort was worse when it occurred in a healthy heart than when it occurred in a heart that was diseased.

Finally, complicating coronary thrombosis a condition of cardiac aneurysm was likely to occur without clinical manifestations, X-ray examination of the heart being often the only means of detecting it in the living subject.

He had published an account of a case in which the patient was an old anginal subject who had had coronary thrombosis with cardiac aneurysm.[1] The size of the aneurysm had been noted by serial skiagrams for nearly two years, following the initial acute attack of coronary thrombosis. It had increased during that period to about six times its original size, and it was surprising to him that, considering this increase, the patient had outlived the acute attack for so long.

If the patients in these cases were subjected to X-ray examination of the heart it would probably be found that cardiac aneurysm complicating coronary occlusion was not uncommon, and he would like to know if Professor Hay thought that such a complication materially affected the prognosis.

1 *Proceedings*, 1932, xxvi, 139 (Clin. Sect., 15); *ibid.*, 1934, xxvii, 1468 (Clin. Sect., 86).

Dr. Plesch: From what has been already said, it is evident that our knowledge of the prognosis of coronary thrombosis is still imperfect. In my view this must continue to be the case until we have more accurate understanding of the causes of angina pectoris, of which coronary occlusion is only one. It is certain that angina pectoris can present most of its characteristic phenomena in the absence of coronary occlusion, as demonstrable at autopsy. If this is allowed, it follows that the prognosis of coronary thrombosis must be separated entirely from the prognosis of angina pectoris. The symptoms of occlusion of the coronary arteries are well known, as are the symptoms of the "status anginosus" of a number of other arteries. We have, however, no knowledge of the pathological conditions of other arteries which might be blamed as being partly or wholly the causes of the non-coronarial forms of angina.

Intercostal arteries.—As every pathologist knows, arterial sclerosis occurs first on the posterior part of the ascending aorta. This is the place of origin of the intercostal arteries, which have some resemblance to the coronaries, as they also supply muscles which work incessantly. As the intercostal arteries supply the intercostal nerves, and some others as well, it seems not unjustifiable to assume that the failure of these arteries accounts for many of the respiratory and sensory troubles in cases of angina pectoris.

Bronchial arteries.—The bronchial arteries are just above the intercostal arteries, and they also are among those most easily affected by sclerosis. They supply the tissues of the lungs, and their occlusion must therefore cause severe disturbance of respiration.

Finally, there are the coronary arteries themselves, and, for the present purpose, these cannot be regarded as a unit. The thrombi occurring in the subsequent anginal attacks do not always obliterate the whole coronary system, but only block out some branches of it, and the consequences of such an occlusion must depend on the place at which it occurs, especially since there are many anastomoses in the coronary system.

The auricular artery.—Most important is the distinction between the occlusion of the first and largest branch of this system—the auricular artery—and the occlusion at any other point. Obstruction in vessels supplying the ventricles is not necessarily followed by death, or even by a severe deficiency of heart function, as these vessels are inter-connected to such an extent as to allow the metabolism of the muscle to continue. On the other hand the auricular artery is a real *end*-artery and therefore deserves more attention, although it has been neglected to some extent not only by pathologists and clinicians, but even by anatomists. Since it is an end-artery, its occlusion involves a complete paralysis of the auricle. This causes among other things an accentuated fluctuation and pressure in the vena cava and the pulmonary veins—leading almost always, in the case of the vein, to œdema of the lungs and death. On these lines we may, even to-day, collate the symptoms of angina pectoris in a causal system.

With help from further researches by pathologists we should in the near future acquire more confidence in the diagnosis and prognosis of angina pectoris, and even obtain further light with regard to its treatment.

Professor Hay (in reply) said, with regard to the prognostic significance of cardiac aneurysm, that the number of cases, more particularly of those which had been watched from the time of the initial thrombosis to that of death, was so small that it was impossible to make any definite statement.

In his opinion the presence of Q wave was of little prognostic value, although it was of some service in substantiating a diagnosis of previous occlusion.

Coronary occlusion was most unlikely to occur in the healthy heart unless it was due to embolism, and the greater the amount of healthy myocardium remaining after the cardiac accident, the better the outlook.

On the general question as to the attitude to be adopted towards inquiring relatives, he thought that during the acute attack one could only talk of possibilities; probabilities might be discussed later on.

He hoped that there would be another discussion by the Section in five years' time, when the whole subject could be approached with greater confidence.

Section for the Study of Disease in Children

President—R. C. JEWESBURY, M.D.

[*October* 26, 1934]

Congenital Obstruction of Œsophagus.—HAZEL H. CHODAK GREGORY, M.D., M.R.C.P., and G. T. CALTHROP, M.D., D.M.R.E.

B. S., male child, aged 12 months, admitted to hospital in August 1934, on account of vomiting and failure to gain weight. Father, Japanese; mother, English. Full-time, normal birth; birth-weight 7½ lb. Breast-fed for three months, then dried milk. Gained weight at first, steadily but slowly (11 lb. 12 oz. at 6 months); then began to lose. Had measles and otitis media at 8 months, at which time weight was 11 lb. 2 oz. Kept at Fever Hospital for four months, during which various foods were given and returned, and was finally transferred to the East London Hospital for Children as a case of obstinate marasmus. His weight was then 10 lb. 6 oz.

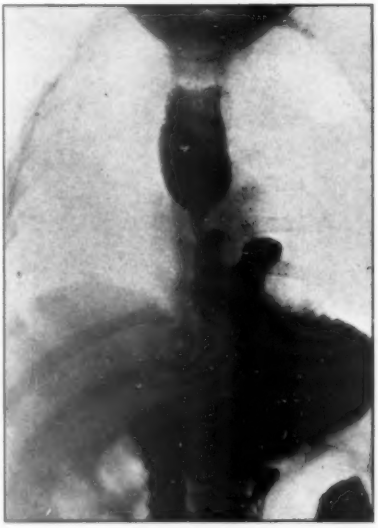


FIG. 1.—Œsophageal stricture. Showing dilatation above the obstruction. The protruding shadow below was seen on the screen to be of a temporary character and capable of enlarging considerably.

Present condition.—A thin, undergrown, intelligent child; height 25 in. Heart, lungs, abdomen, throat, ears, and urinary system normal. No other congenital abnormalities present. Fluid is generally, though not always, swallowed and retained; solids are invariably regurgitated soon after swallowing. Salivation is increased and the saliva is regurgitated in large quantities. X-ray examination of the chest reveals no abnormality. The diaphragm moves well and equally on each side. When one mouthful of barium meal of fluid consistency is given, it is seen to stop at the level of the seventh rib posteriorly. Above this point the œsophagus is dilated. When more meal is swallowed, it is seen to pass through a narrow constriction about ¼ in. in length. Distal to the constriction, the œsophagus is of normal width,

but at times there is seen a bulging or branching to the left, well above the level of the diaphragm. When the child cries, the stomach appears to be squeezed out of the abdominal cavity, and the bulge spreads until it reaches the outer thoracic wall. As soon as the cry stops, the stomach slips back again into the abdomen. The meal then passes on without any other evidence of abnormality.

Since September 10 a sodium bicarbonate wash-out of the œsophagus has been carried out twice daily, and this has been followed by considerable improvement. Large quantities of mucus are evacuated by the lavage. Even with this treatment the child suffers from severe exacerbations in which he returns even fluids, and loses as much as 12 oz. in a day; he recovers however always slightly more than

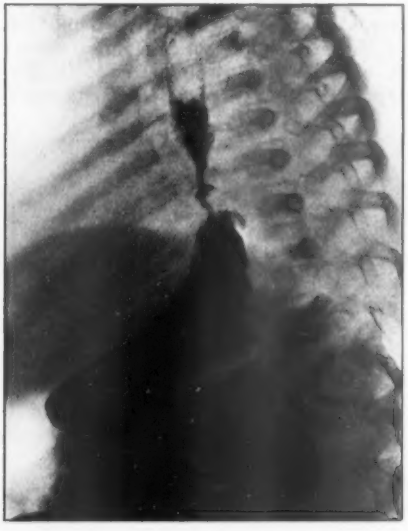


FIG. 2.—Lateral view of œsophageal stricture.

he has lost, so that the general tendency at present is in an upward direction. He is being fed on milk, Benger's food, ovaltine, soup, and orange-juice. On October 11, after a bad week of regurgitation and loss of weight, he vomited a solid compact mass the size of a haricot bean, which consisted of compressed milk curd. It had probably been acting as a ball-valve, and was responsible for the recent relapse and loss of weight.

As the child is making slow but certain progress on a fluid diet, and as a study of the literature shows, on the whole, very poor results from operative interference, it has been decided to send him to Chailey to be nursed in the open air, away from infection, and to continue conservative treatment in the hope of spontaneous cure.

Discussion.—Dr. W. M. FELDMAN asked whether an œsophagoscopic examination had been made to ascertain whether the obstruction was fibrous or membranous, because in the latter case perforation or stretching of the membranous diaphragm under inspection might result in a cure.

Dr. HELEN MACKAY said that a child—now over 2½ years old—with similar radiographic findings, due apparently to a short œsophagus and a partially thoracic stomach, had been attending the Queen's Hospital for Children since he was two months old. As in other such cases, the predominant symptom was vomiting, of varying severity, and the vomit sometimes

contained altered blood. This child had been shown by Dr. Ursula Shelley at the International Pædiatric Congress in 1933, and she (Dr. Mackay) understood that the consensus of opinion among those familiar with such cases was that operative interference was not indicated. The small boy in question was still fed almost entirely on fluids. His general condition was good, though he was very small for his age.

Congenital Elevation of the Diaphragm.—T. STANLEY RODGERS, M.D. (for A. G. MAITLAND-JONES, M.D.).

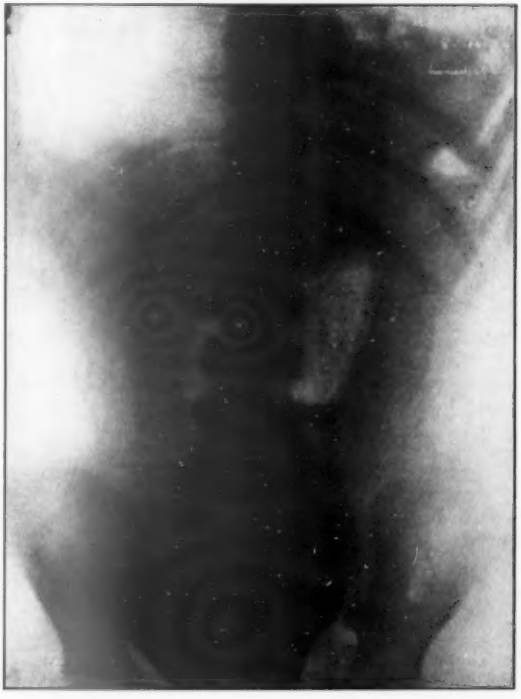
L. H., female, aged 5 years, was brought to the London Hospital in July 1934, complaining of a chronic cough which she had had for some months.

On examination.—Small for her age, pigeon-chested and poorly nourished. At the left base the percussion note is a little impaired and the breath sounds are diminished. There are no other abnormal physical signs.

Radiography.—Screen examination showed that the left half of the diaphragm was raised considerably and only moved slightly with respiration. There was no paradoxical movement. A skiagram taken after the ingestion of bismuth showed that the stomach lay just below the left cupola of the diaphragm. Cervical ribs and, on the right side, an azygous pulmonary lobe were also present.

Diaphragmatic Hernia.—R. W. B. ELLIS, M.D. (by permission of H. THURSFIELD, M.D.).

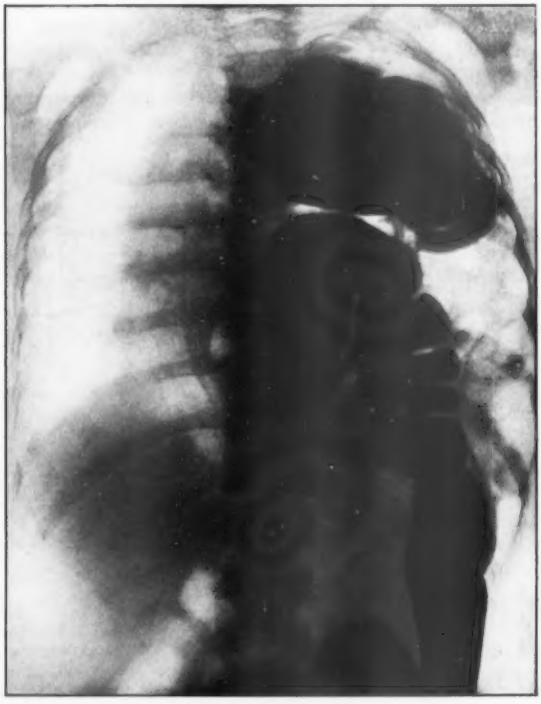
R. E., male, aged 6½ years. One older brother and both parents healthy. Birth-weight 9 lb.; normal labour; "some difficulty in getting child to breathe." There have been no subsequent attacks of cyanosis. The boy had measles and chicken-pox in infancy, and bronchitis each winter, but has otherwise been in good general



Diaphragmatic hernia Bismuth meal. Fundus of stomach within pelvis.

health until six months ago. Since this time he has complained occasionally of abdominal pain (never severe), referred to the umbilicus, and has vomited nearly every evening. The vomiting occurs after he has gone to sleep, usually about 8.30 p.m., and appears effortless and unaccompanied by nausea. The vomitus consists of food eaten throughout the whole day.

On examination.—Healthy-looking, active boy, weighing 39 lb. No deformity of chest, though there is diminished movement on the left. Heart displaced to right (area of cardiac dullness extends one inch to right of sternal border). Percussion note tympanitic (showing considerable variation) over left lower chest; no impairment of percussion note on right. Breath sounds greatly diminished over left lower



Diaphragmatic hernia. Barium enema. Cæcum and colon in left hemithorax. Heart displaced to right.

chest, anteriorly and posteriorly, with moderately good air entry at apex. Borborygmi occasionally heard on left. Abdomen: slight fullness above pubis; no other abnormality. No tenderness.

Barium meal shows a greatly dilated stomach, with the fundus lying at the level of the pelvic brim. The cardiac orifice is normally situated. Barium enema shows the sigmoid, transverse colon, and (?) cæcum lying within the left chest.

Discussion.—Mr. L. E. BARRINGTON-WARD said that he had been asked to see this case in the ward by Dr. Thursfield. He was of opinion that an operation should be attempted for the repair of the hernia. If no operation were done, intestinal obstruction was very

likely to occur sometime. He agreed that the gap might be a large one, but he thought that it might be possible to close the opening by a fascial graft, if other methods failed.

The PRESIDENT referred to a case of congenital diaphragmatic hernia in an infant aged 4½ months. X-ray examination had shown the left pleural cavity to be filled with intestine. In this case an operation was performed by Mr. Max Page. The abdomen, opened by a left paramedian abdominal incision, appeared to be practically empty; all the abdominal viscera except the stomach had passed into the thorax. The herniated bowel was reduced by gentle retraction from below without very much difficulty, and the opening in the diaphragm was closed with sutures. The child's condition improved very much after the operation, but unfortunately, about three weeks later, an acute intestinal obstruction developed and proved fatal.

Post mortem, it was found that the orifice in the diaphragm had been successfully closed; the mobility of the intestines, however, was such that a portion of the small bowel had undergone rotation around an adhesion and had become gangrenous.

Some cases of diaphragmatic hernia are curable by operation, particularly those occurring on the left side and approach through the abdomen rather than through the thorax is the method to be preferred. Treatment by operation ought always to be considered carefully. He (the President) did not think that the present case was suitable for operation, as there appeared to be almost a complete absence of diaphragm on the left side.

Neoplasm of Lung. (? Teratoma).—R. W. B. ELLIS, M.D. (by permission of H. THURSFIELD, M.D.).

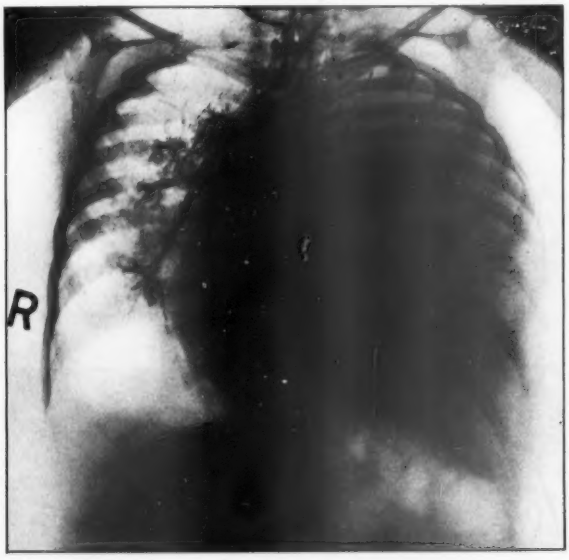
J. S., female, aged 4½ years. Only child; parents healthy. Normal infancy. The child was well until age of 2 years, when the abdomen began to swell and there was frequent diarrhœa. She was admitted to the Hospital for Sick Children, Great Ormond Street, in November 1932, aged 2 years 8 months. She then weighed 26 lb.; the abdomen was distended, the liver palpable two fingerbreadths and the spleen one fingerbreadth below the costal margin. The chest was deformed, with enlargement of the left side and distension of the superficial veins.

23.11.32.—Tumour occupying lower two-thirds of left chest, with well-defined upper margin.

The rib spaces were filled on the left and the left chest was dull to percussion front and back, though resonant above the clavicle. No breath sounds were heard over the left lung.

Radiological examination (25.11.32).—The right diaphragm moved normally; the left base showed a dense shadow with a well-defined upper margin, which failed to move on respiration. A barium meal showed the œsophagus displaced to the right, but the stomach normally situated. There was no delay in the passage of the meal. The chest was needled in the ninth space posteriorly; no fluid obtained. Needle appeared to be entering solid lung. No cells seen in content of needle.

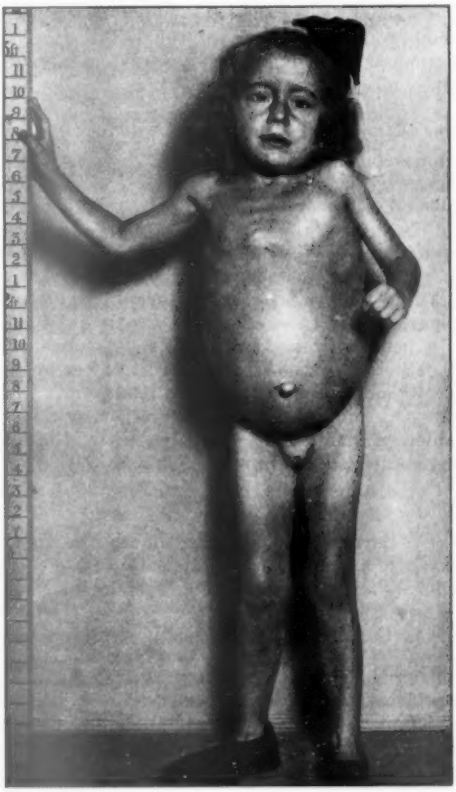
The child was admitted to the Royal Hospital for Sick Children, Glasgow, for two months in 1932, under Professor G. Fleming, and attended there during the following two years. The chest was needled several times, 22 c.c. of bloody fluid



28.9.34.—The tumour now occupies almost the whole of the left chest, and displaces the mediastinum to right. Lipiodol shows extreme deflections of trachea to right, and compression of right lung, but has only entered ½ in. of left bronchus.

being withdrawn on the first occasion. A small amount of blood subsequently obtained was hardened and cut by Dr. Blacklock, who reported: "A few clumps of large cells with granular protoplasm and large darkly staining nuclei are found. The cells are very suggestive of some malignant condition." Whilst attending under Dr. Fleming in September 1933, the child began to complain of attacks of pain in the back, and the radiologist reported "a suspicious area, lower dorsal vertebræ." Physical examination did not reveal any spine lesion. During January and February 1933 she had deep X-ray therapy which did not appear to have any influence on the condition. The abdomen has been greatly distended throughout the past two years, though the distension is less when she is running about. Barium enema showed a greatly distended colon and sigmoid. Her general health is otherwise moderately good, and she has gained weight slowly.

On examination.—Pale, rather frail child, not obviously wasted. Weight 39 lb. Tendency to dyspnœa on exertion; no cyanosis or clubbing. Trachea displaced far to right. Pigeon-breast deformity of chest, with bulging of the whole of the left side, and filling of the left intercostal spaces. The area of cardiac dullness extends two fingerbreadths to the right of the sternal border. Heart sounds best heard half an inch internal to the right nipple line. Diminished movement and stony dullness to percussion over left chest. Sonorous rhonchi over right lung; breath sounds



Neoplasm of lung: showing deformity of chest and abdominal distension.

entirely absent over left chest, and no adventitious sounds heard. Abdomen very considerably distended, with small umbilical hernia. No free fluid. Liver two fingerbreadths and spleen three fingerbreadths below costal margin. No adenopathy.

Wassermann reaction negative. Mantoux test negative.

Radiological examination (Dr. Shires, 28.9.34) shows great displacement of heart and mediastinum to right, and opacity of whole of left lung. Lipiodol injected by the cricothyroid route, shows the trachea bent sharply to the right, and the right lung occupying a relatively small part of the right chest. The oil fails to pass more than half an inch into the left bronchus.

Giantism, Virilism, and Pseudohermaphroditism. — J. VERNON BRAITHWAITE, M.D.

Irene C., aged 4 years, admitted 24.6.34 on account of overgrowth, genital precocity, and deepness of the voice. At birth there was some doubt as to the sex, but it was decided that the child was a girl. For two years, although she was a large child, the mother did not think that she was abnormal. At this time, however, the genitals became enlarged and pubic hair appeared. Since then the condition has gradually increased. The voice became loud, and then deep. She has been to school for a year and is apparently quite intelligent.

Father died of embryonal carcinoma of the left testicle in January 1933. A brother died of pneumonia at 3 months. Two sisters, aged 7 years and 5 months respectively, alive and well. Mother healthy.

Weight record for first two years

Age	Weight
6 weeks	8 lb. 15 oz.
6 months	17 lb. 10 oz.
12 months	25 lb. 10 oz.
18 months	32 lb. 8 oz.
2 years	36 lb. 0 oz.

Condition on examination.—Weight 58½ lb. General appearance that of a child about twice her age. No hair on face. Hair on head fair. There is a compact patch of long, dark pubic hair, with a masculine distribution. The genital organs have a female configuration, but the clitoris is greatly enlarged, and is comparable in appearance with a penis with a ventral cleft. At the base of this organ is a single passage. A catheter passed through this, after encountering an elastic obstruction about two inches from the orifice, passes into the bladder. There is no sign of a vagina. On bimanual rectal examination under anæsthesia with a catheter in the bladder, a small mass is palpable in the position of the cervix above and behind the catheter. No prostate could be felt.

Investigations.—Urine, no abnormality. Basal metabolic rate, plus 10%. Blood-sugar curve normal.

Skiagram of skull.—Beyond a tendency to thumbing and some approximation of the clinoid processes, there are no abnormal appearances.

No abnormal signs in the central nervous system, and no palpable abdominal tumour.

Suggested diagnosis: Congenital hyperplasia of suprarenal cortex.

Discussion.—Dr. PARKES WEBER said that when suprarenal cortical adenoma (or hyperplasia) caused pseudohermaphroditism as well as virilism in females, the disease must be congenital, as in the present case. The foreign term "inter-renalism" was convenient, the inter-renal organ of some lower animals being represented by the suprarenal cortex in human beings.

Dr. R. W. B. ELLIS compared this case with a post-mortem specimen shown at a meeting of the Clinical Section on December 8, 1933,[1] in which great hypertrophy of the adrenal glands had been associated with the presence of ovaries, uterus, a vagina ending blindly above the perineum, a scrotum, and a penis with the urethra opening at the base of the glans. (The ovaries had at the time been reported as being of normal structure, but further examination, undertaken at the suggestion of Dr. Levy Simpson, had shown the presence of rete testis within them.) In that instance the infant appeared to have been a female, the external genitalia having been converted almost perfectly into those of a male—even to the extent of the urethal orifice being situated near the distal end of the clitoris-penis. This conversion to the male type must have occurred *in utero*, since the external genitalia were in their present state at birth.

[1] *Proceedings*, 1934, xxvii, 401 (Clin. Sect., 39).

Precocious General Development and Obesity.—F. PARKES WEBER, M.D.

R. G., female, aged 13½ years. A big fat girl, with a good deal of pubic and axillary hair, and with fine "striæ atrophicæ" over both hips. Height, 148 cm.; body-weight, 67·1 kilograms (10 st. 8 lb.) By radiograms of the hands Dr. Wood reports that there is union of epiphyses in all the phalanges—corresponding to the age of 17 years. Nothing else noteworthy by general examination. No abdominal tumour felt. Urine, free from albumin and sugar. Brachial blood-pressure, about 100 mm. Hg (systolic). Thyroid gland not enlarged. The pituitary fossa appears normal by X-ray examination. The Wassermann reaction is negative.

According to the mother the patient has always been a "big girl," but has become increasingly fat during the last one and a half years. Menstruation commenced sixteen months ago and has been regular since then, sometimes very abundant. The child's behaviour and mental development are normal, but (according to the mother) she often seems tired and without energy. There is no family history of importance.

I think that the tendency to obesity is of endocrine origin, but the deviation from normal development hardly amounts to disease. I have seen similar precocious osseous development, associated with precocious general development, in other girls, in whom the deviation from normal growth hardly amounted to disease. From one point of view I feel inclined to regard many cases of rapid general development[1] in children (and some cases of hirsuties), as representing a kind of reversion to a lower type. Primitive man had probably to develop more rapidly in order to survive, though ultimately the individuals of relatively slower general development have proved themselves the fittest to survive.

Hydrocephalus with Precocious Puberty following Post-basic Meningitis.—BERNARD SCHLESINGER, M.D.

Patient is a girl now aged 8 years 8 months.

History.—She is one of twins, both of whom had post-basic meningitis when four months old. Her sister died and the surviving twin developed a progressive hydrocephalus since that illness. When first seen in June 1930, she was suffering from fits, hydrocephalus and signs of spastic paresis, with bilateral extensor responses, adductor spasm, and right facial weakness. She was unable to sit up by herself or to stand. The discs appeared to be normal. The Wassermann reaction was negative. During the next two years her condition did not alter appreciably but the fits disappeared under treatment by sodium luminal. In 1932 she began to sit up and a little later was able to stand and to make poor attempts at walking with help. In 1933, at the age of 7, the breasts began to develop. A skiagram of the skull showed the pituitary fossa to be within normal limits and the calvarium to have a beaten silver appearance. A blood-sugar curve (Dr. Payne) following 26 grams of dextrose, showed a total rise on the low side of normal (0·100%, 0·164%, 0·120%, 0·093%, 0·067%) at the end of three hours. Recently the sexual development has slowly increased, but as yet there has been no menstruation.

The optic discs are now becoming pathologically pale, and there are signs of primary optic atrophy (Mr. G. G. Penman).

The patient's mentality has always been somewhat peculiar. She is certainly mentally retarded, has a habit of reiterating things one says to her and for no known reason will suddenly burst into bad, abusive language.

Dr. PARKES WEBER said that it was relatively rare to see chronic internal hydrocephalus following basal meningitis, but hardly anyone had seen a case of the kind accompanied by precocious puberty; nevertheless, this association had been described. Cases of so-called pineal precocity (or "pineal gigantism") occurred almost only in male children.

[1] For instance, two cases that I have previously demonstrated (*Proc. Roy. Soc. Med.*, 1933-1934, xxvii, pp. 589 and 637), in both of which there was similar precocious osseous development to that in the present case.

Congenital Recto-urinary Fistula in a Girl.—D. MAKEPEACE, M.D. and MARY GIBSON, L.R.C.P., M.R.C.S. (for HAZEL CHODAK GREGORY, M.D.).

S. H., female, aged two months. Born in the Mothers' Hospital. The mother was a primipara, aged 32, who had a normal pregnancy and labour. Birth-weight 8 lb. 2 oz. During first nine days nothing abnormal was noted about the baby, except a failure to gain. The weight remained about 7 lb. 11 oz. in spite of supplementary feeds, there being very little breast milk.

10th day: 6 a.m., profuse sweating and very rapid respirations. Temperature 106·8° F., nothing abnormal found on examination. Nine ounce loss of weight in twenty-four hours. During the day the baby appeared better; took feeds well. It was noticed that napkins were wet further back than normal. At 6 p.m. temperature 101° F. Fluid collected as passed per rectum contained 0·45% urea. A normal stool followed immediately, no urine seen coming from urethra. Pot cit. gr. iv, given 4-hourly.

11th day: Temperature fell to normal. Five-ounce gain in weight. Appeared to be well. Urine passed per urethram, stools very fluid.

12th day (September 6): Transferred to Royal Free Hospital for further investigation.

Condition on admission.—Temperature 98; pulse 120; respirations 38. Weight 7 lb. 10 oz. Fluid was seen to pass from the rectum and from the urethra but none from the vagina.

On examination.—Genitalia appeared to be normal. Perineum very short. Small depression in the midline over the coccyx. No spina bifida, hare-lip, cleft palate, or other congenital abnormality seen. No evidence of a congenital cardiac lesion. All systems: nothing abnormal discovered.

Rectal examination: (1) Digital: Nothing abnormal felt; (2) Sigmoidoscopy: A (?) small opening was seen on the anterior wall of the gut 2 in. above the anal orifice. It proved impossible to pass a probe through the opening.

Vaginal examination: A probe was passed but nothing abnormal could be felt.

Radiological examinations: (1) Uroselectan—2 minutes, uroselectan in pelvis of kidney; 5 minutes, ? uroselectan seen in rectum; 15 minutes, uroselectan seen in pelvis and bladder; 25 minutes, uroselectan seen in pelvis and bladder; 35 minutes, uroselectan seen in pelvis and bladder. At no time were the ureters seen. (2) X-ray examination of spine: no spina bifida seen. First sacral segment appears to be irregular, but may be due to gas.

Pathological examination: (1) Fluid from the urethra (washed-up specimen). Pale, slightly turbid. No albumin, blood, sugar, acetone bodies, bile-salts or pigments. Microscopically (centrifuged specimen): Many epithelial cells, some pus cells and a few red blood cells. Culture: Coliform bacilli and streptococci isolated.

(2) Fluid from rectum: Turbid, slightly darker than that from urethra. Urea present.

General Progress.—The baby was kept on potassium citrate, gr. iv t.d.s. throughout her stay in hospital. She took her feeds of citrated milk well and gained weight. On discharge nine days later she weighed 8 lb. 4 oz.

She has since been seen from time to time and seems to be in good health, is contented, and is gaining weight; she still passes urine from both the rectum and the urethra.

Hepatomegaly. ? V. Gierke's Disease.—PHYLLIS WATSON, L.R.C.P., M.R.C.S. (for Dr. ALICE KING).

Leonard Drake, aged 3 years.

History.—Brought to the Queen's Hospital Outpatient Department (under Dr. Alice King) on account of enlargement of the abdomen, noticed by the Welfare Centre in May 1934.

The only complaint of the mother was that the size of the abdomen impeded the child's walking. Apart from occasional bronchitis he was healthy, active, and cheerful, with good appetite.

The child was full-term, the labour normal; birth-weight 8 lb. 8 oz.; breast-fed until three months, then had dried milk feeds until on solid diet. Present diet normal for his age.

Admitted 31.7.34 for investigation. Complete physical examination revealed no abnormal physical signs except the abdominal enlargement, the girth being 21 in. (constant throughout stay in hospital).

The liver edge was three fingerbreadths below the costal margin, the right lower edge being about 1 in. above the right iliac crest. The enlargement was regular, the surface smooth. The costal angle was wide. The spleen was not palpable. There was no free fluid. The bowels were regular, rather loose; motions otherwise normal.

Family history.—Father and mother both healthy; sister, aged 4 years, healthy; showed no enlargement of liver; brother, aged 1 year, healthy, and also showed no enlargement of liver.

Investigations.—Urine examined repeatedly. Occasionally trace of acetone. No other abnormality. Wassermann reaction negative 2.8.34.

X-ray examination: Long bones. No active rickets seen. Abdomen: large liver shadows; no cysts.

1.8.34: Blood-glycogen 17·6 mg.%.

10.8.34: Van den Bergh (direct and indirect tests) negative.

16.8.34: Stool: no parasites seen. Benzidin test weakly positive.

8.8.34: White blood-cell count 19,200 per c.mm. *Differential:* Polys. 23%: lymphos. 64%; eosinos. 13%.

30.8.34: Differential white-cell count: Polys. 24%; lymphos. 69%; eosinos. 7%.

5.9.34: Whole-blood cholesterol: 132 mgm. per 100 c.c.

Blood-sugar, 29.8.34:—

Resting		0·079	10 a.m.	
			10.15 a.m.	Urine, no acetone
After 30 grm. glucose	½ hr.	0·134	10.30 a.m.	
	1 hr.	0·152	11 a.m.	Urine, acetone +
	1½ hr.	0·131	11.30 a.m.	
			11.45 a.m.	No acetone
	2 hr.	0·113	12 noon	
Resting		0·070	10 a.m.	
	½ hr.	0·131	10.30 a.m.	Trace acetone in urine
After adrenalin ♏ iii	1 hr.	0·092	11 a.m.	
	1½ hr.	0·074	11.30 a.m.	
	2 hr.	0·066	12 noon	Trace acetone in urine

Resting	10 a.m.	0·088
After 30 grm. lævulose	10.20 a.m.	0·113
	10.40 a.m.	0·102
	11 a.m.	0·092
	No acetone in urine	

? Idiopathic Hypertrophy of the Heart.—A. M. NUSSBRECHER, M.D. (by permission of Dr. ALAN MONCRIEFF).

S. C., female, aged 16 months. Parents not related. Mother had swollen legs and "sickness" when carrying patient. Father suffers from epileptic fits and has a "bad heart." There are two other children by the mother's first husband, both healthy.

Full-time baby, 8 lb. at birth. Breast-feeding for two months, later supplemented by milk and water; since five months old on bottle only. Gained well and at a year weighed 22 lb. Bullous impetigo at 9 weeks; transient diarrhœa and vomiting associated with mother's medicine at 3 months; otitis media at 6 months followed by diarrhœa and vomiting, the otitis continuing intermittently; slight bronchitis at times.

Sat up at 6 months; walked around objects since a year old. Never appeared in pain, apart from earache. Never blue. Rapid breathing noticed for about two weeks before admission to the Middlesex Hospital under Dr. A. Moncrieff, where she was referred because of a cough and loss of weight.

On admission (1.9.34).—Pale and listless. Temperature (rectal) 99·2; pulse 130; respirations 40; Weight 20 lb. 4 oz. Anterior fontanelle just open. Twelve good teeth. Tonsils moderately enlarged; a few small glands in the neck. Heart regular. Apex beat in fifth space, almost in anterior axillary line, forcible. Second aortic sound accentuated; otherwise nothing abnormal found on auscultation. Blood-pressure 110/80. Veins in neck prominent. Liver one-and-a-half fingerbreadths down; spleen not felt. Lungs clear. Ear-drums normal.

Investigations.—X-ray (fig. 1): Heart enlarged in all diameters. Cardio-thoracic-ratio: 0·84. Electrocardiogram (fig. 2): "Normal rhythm; rate 125. The T waves

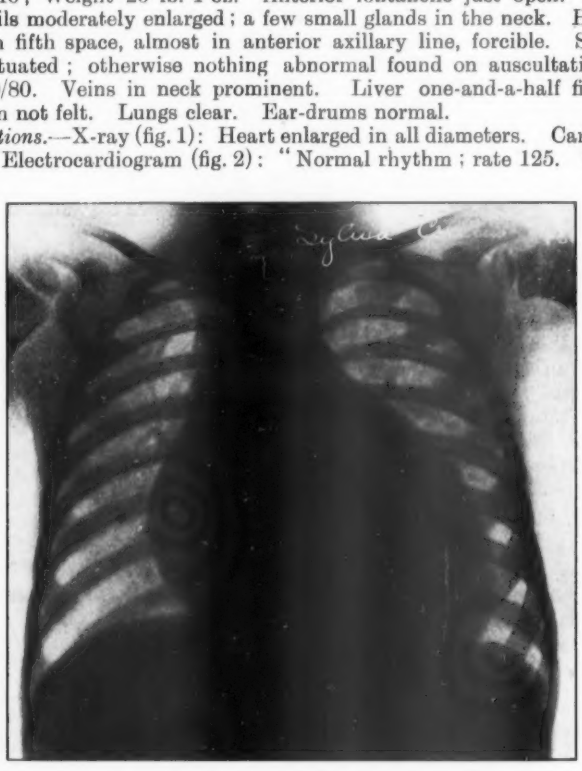


FIG. 1.—Antero-posterior view showing marked increase in transverse diameter of heart.

are inverted in lead I; P waves notched in all leads. The curve indicates an abnormal myocardium." (Dr. Evan Bedford's report.)

Blood-count.—Hb. 90%; R.B.C. 5,340,000; W.B.C. 15,600. *Differential*: Polys. 47%; lymphos. 40%; monos. 13%.

Blood-urea 33; non-protein nitrogen 30.

Urine normal. Catheter specimen: No deposit; sterile.

Mantoux test negative in 1 : 10,000, and 1 : 1,000. Wassermann reaction negative.

Progress.—Stayed in hospital till 20.9.34 and during that time was practically afebrile, average respiration rate about 44; average pulse-rate 130. Two further readings of blood-pressure were both 120/80. General condition unchanged.

Given tinct. digitalis ♏ 5 t.d.s. from 16.9.34, increasing to ♏ 6 t.d.s. on discharge.

This was soon followed by vomiting and diarrhœa and the drug had to be stopped. When seen on October 10, patient appeared to be very much better and

her mother stated that she was now full of life and that her appetite had returned. There had been a gain of 2 lb. in two weeks. Whereas previously she had been listless, and did not object or even cry much at venepuncture or taking of blood-pressure, she now yelled and threw herself about to such an extent that a blood-pressure reading or pulse-taking became difficult and unreliable (respective figures: 130/90 mm. Hg and 144). Respiration rate 44.

The outstanding features of the case are: (1) The apparently insidious onset, (2) the enlargement of the heart, (3) the markedly raised blood-pressure, with evidence of good circulation in the lower extremities, (blood-pressure in legs 110 syst.), (4) the abnormal electrocardiogram, (5) the absence of other obvious causes of tachycardia and tachypnœa, and (6) the fact that though the last two symptoms have persisted, the general condition has improved spontaneously.

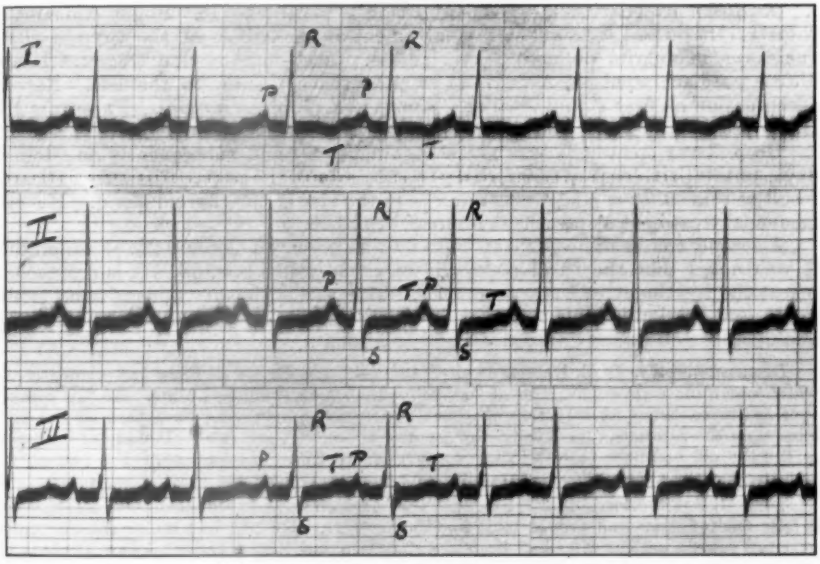


FIG. 2.—Electrocardiogram showing invertion of T s in lead I and notchings of P s in all leads.

The child is shown more for diagnosis than as an actual cast-iron case of idiopathic hypertrophy of the heart, in the sense used by Howland and others.

At first glance pericarditis with effusion has to be considered, but the forcible apex-beat, displaced well to the left, and the X-ray appearances are definitely against this supposition.

The most likely condition is undoubtedly some form of congenital heart disease, possibly an early coarctation of the aorta. The absence of murmurs and of other signs apart from the enlargement of the heart does not militate against such a diagnosis.

Dr. N. B. CAPON referred to an example in which congenital narrowness of the aorta at the point of attachment of the obliterated ductus arteriosus was believed to be the cause of the hypertrophy; the late Dr. Carey Coombs, who saw the post-mortem specimen, agreed with this view. The left coronary artery arose from the pulmonary artery, but a study of the literature suggested that this was unlikely to have been the cause.

Graves' Disease in a Child aged 8½ years.—H. E. MANSELL, B.M.

John B., admitted to St. George's Hospital, January 3, 1933, at the age of 6 years 9 months, suffering from facial impetigo and cervical adenitis. Discharged on January 18 to a convalescent home, where, a few weeks later, he was noticed to have a tachycardia of 120-140, enlargement of the thyroid gland, slight prominence of the eyes, and loss of weight in spite of a voracious appetite. He was treated with rest, glucose (¼ lb. daily), and liq. iodi mitis ℿ 5 t.d.s. Discharged home in June, but readmitted to hospital a month later. Medical treatment was continued on similar lines, with the addition of insulin, 6½ units b.d. He was eventually discharged to Queen Mary's Hospital, Carshalton, on April 18, 1934, weighing 4 st., and with his pulse-rate reduced to 90-120. After two months there he was transferred to the special clinic at New End Hospital, and thence on August 18 to Lambeth Hospital for X-ray therapy.

Condition on admission.—Good nutrition. Weight 58 lb. Slight widening of palpebral fissures. General soft enlargement of the whole of the thyroid gland. Increased manubrial dullness 2¼ in. across. Well-marked fine tremor. Heart: diffuse cardiac impulse. Apex beat in fifth space, 3½ in. to the left. Nervous accentuation of first sound at apex. Lungs and abdomen normal. Pulse-rate 138. Blood-pressure 148/68. Basal metabolic rate (by Reid's formula) + 72.

Deep X-ray treatment was given on August 30 and subsequently on alternate days, to total six doses of 150 *r*, three to each side of the neck. The only noticeable result of this so far is a slight diminution in the size of the thyroid gland. His only medicinal treatment at present is luminal, gr. ½ b.d.

A second course of deep X-ray therapy was begun on October 23, at which time it was noted that the basal metabolic rate had fallen to +58. It is proposed to repeat the treatment every two months. The case is shown as one of considerable rarity, and opinion is sought upon the possible relation of onset to previous infection and upon the questions of prognosis and treatment.

Discussion.—Mr. L. E. BARRINGTON-WARD said that Dr. R. S. Frew had shown a case which was a more severe example of Graves' disease than the present one. He (the speaker) operated three times at intervals of about two months, removing first, part of the right lobe, then the isthmus, and thirdly, the greater part of the left lobe. It was not until most of the thyroid had been removed that improvement set in. He had seen the patient this summer and she appeared to be cured in every respect.

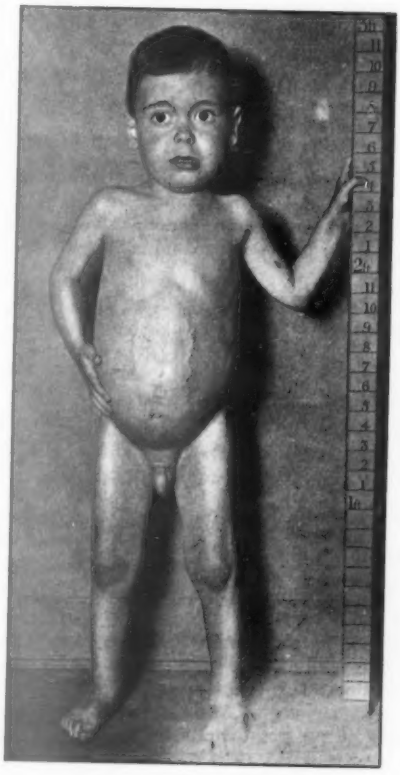
Dr. F. J. POYNTON said that the earliest age at which he had seen the disease was 2½ years; it followed influenza and proved fatal in the fifth year. He had had under observation for over ten years a patient who had been at Great Ormond Street under his care at about the age of 10 years with very definite signs—large thyroid, projecting eyes and tachycardia. She had made a good recovery, but the prominent eyes were still obvious. This patient had been treated with X-rays during childhood. He was not quite sure that with the advance in surgery if he had such a case again he would not prefer operation, but this was clearly a matter of opinion.

Cœliac Disease with Unusual Features.—P. R. EVANS, M.B. (for HUGH THURSFIELD, D.M.).

J. T., male, aged 4 years. Healthy family, two sisters, aged 9 and 7, and one twin brother. Pneumonia at 5 months, followed by repeated attacks of bronchitis. Pneumonia again at 17 months, after which the child stopped growing, stopped walking, passed 4 to 5 large, slimy, unformed, greenish-yellow, foul stools every day, and had occasional attacks of vomiting lasting one day; excellent appetite; large abdomen, with veins in the wall which became gradually more obvious; slight cough.

On admission (August 24).—Pale; could not stand. Teeth good; tonsils and cervical glands enlarged. No abnormal signs in lungs, but clubbing of fingers, fairly

frequent cough, respirations 24/min. Systolic murmur maximal in pulmonary area, no other abnormal cardiac signs, pulse-rate 100. Abdomen large and flabby, no mass felt, no ascites; six tortuous veins on anterior abdominal and thoracic walls with blood flowing upwards, apparently from hypogastrium; enlarged vein running out from right groin. Reflexes normal. Mentality good. No pyrexia. Urine normal. Weight: August 24, 25 lb. 12 oz.; October 10, 28 lb. 14 oz.



Cœliac disease: showing distended abdominal veins.

Other investigations.—Stool (August 25): Split fat 35·9 } % of dried fæces.
Unsplit 8·7 }

Or, of fæcal fat 80·4% split.
19·6% unsplit.

Microscopically: excess fatty acid crystals and fat globules, slight excess of starch.

Wassermann reaction negative. Mantoux 1 : 1,000 negative.

Blood-count	Aug. 24	Sept. 10	Sept. 25	Oct. 5
R.B.C.	2,910,000	3,900,000	4,100,000	5,100,000
Hb.	31%	45%	67%	65%
Colour-index ...	0·5	0·57	0·81	0·63
Reticulocytes ...	1%	2·6%		
W.B.C.	9,700	12,400		
Polymorphs ...	42%			
Lymphocytes ...	40%			
Monocytes ...	3%			
Eosinophils ...	3%			
Basophils ...	2%			

Lævulose tolerance:

Blood-sugar before 18 grm. lævulose ...	0·101%
½ hour after	0·109%
1 hour	0·130%
1½ hours	0·128%
2 hours	0·110%

Skiagrams.—Chest: No obvious rickets; root shadows increased. Screening: both sides moved well. Lipiodol: No bronchiectasis. Wrists: Healing rickets.

Barium enema: Dilated atonic colon, consistent with cœliac disease.

Treatment.—Fat-free diet; haliverol; marmite; ferrum redactum, gr. iii, t.d.s., p.c.

Pulmonary Abscess. Recovery.—E. HINDEN, M.R.C.S. (for Dr. DONALD PATERSON.)

G. W., male, aged 18 months, brought to Dr. Paterson's clinic on May 23, 1934. Weight then 23 lb. As he had not been doing well, his parents had taken him some three months previously to King's College Hospital, where he had had his tonsils removed, one week before he was brought to Dr. Paterson. The parents stated definitely that the cough and general condition had been much worse since the operation.

On examination the child was listless and looked ill. Sloughs were present on the tonsillar beds; there were clicking râles over both lungs; the temperature was 99·4. A skiagram showed a shadow in the right upper lobe, with the appearance of ? cavitation ? localized pneumothorax, in the same situation. As there were no vacant beds, the child was admitted to the Elizabeth Garrett Anderson Hospital, under the Hon. Mrs. Olivier Richards, to whom I am indebted for permission to show the in-patient notes and skiagrams.

He was very ill at first; the temperature was swinging, and on one occasion reached 105. The Mantoux reaction was positive, though I could not confirm this finding later; there were no tubercle bacilli found in the abundant sputum. Hæmolytic streptococci were cultured from the sputum, from throat-swabs and from an ear discharge which developed later. The leucocyte count was 30,600 on June 4, and 37,600 on June 14. After June 22 the temperature did not rise above 99·6, and the sputum diminished in quantity. The child contracted a ward infection of chicken-pox, and was discharged home much improved on July 5.

When seen on August 4, he was still coughing a little, but looked much better and livelier; his weight was then 28½ lb. A series of skiagrams demonstrated consolidation and cavitation of the right upper lobe, which cleared up leaving a little shadowing in that lobe, probably due to residual fibrosis. The child is now well, but still coughs at times. There are now no physical signs in the chest.

It has been suggested that a diagnosis of localized pneumothorax would also be compatible with the X-ray findings, but I think that the clinical course almost rules this out.

Congenital Deformities. — E. HINDEN, M.R.C.S., for Dr. PEARSE WILLIAMS.

J. L., female, aged 10 months. This child shows the following abnormalities: Asymmetry of the skull; atrophy of the right pectoralis major; laminæ of the cervical spine not fused; congenital stridor; mediastinal tumour, probably a dermoid; blindness of the right eye, due to a coloboma of the right optic disc.

Still's Disease with Erythema Multiforme.—R. H. BAILEY, L.R.C.P. M.R.C.S. (for Dr. DONALD PATERSON).

J. A., male, aged 6½ years.

History.—Tonsils removed April 1933. In September 1933 had an "urticarial rash" all over the body—most marked on the limbs—pains in the joints, pyrexia and profuse sweating.

On examination.—January 1934: Pale, good-tempered. Apex beat in fourth space in nipple-line; faint systolic bruit. Spleen palpable one fingerbreadth below costal margin. Peri-articular swelling of knees, with slight effusion. Other joints normal.

February 1934: Joints of fingers of both hands swollen. Inguinal and axillary lymph-glands enlarged. Each night a mild erythematous rash appeared on the arms; this had faded by the morning.

Investigations.—Blood-count (29.1.34): R.B.C. 4,200,000; Hb. 80%; W.B.C. 15,000; polys. 81%; lymphos. 15%; monos. 4%. Mantoux 1:100 strongly positive. No tubercle bacilli in stomach washout; no result on injecting this into a guinea-pig. Wassermann reaction normal.

X-rays: No bony changes; enlarged mediastinal glands.

Treatment.—Four series of injections of "halmo-protein," in increasing doses, beginning with 0·1 c.c. and working up to 0·8 c.c.

Sent to the country March 1934.

Readmitted July 1934 with a relapse. Spleen now three fingerbreadths below costal margin. Joint swellings increased.

Dermatological report (Dr. H. T. Barron): "The macular and maculo-papular lesions, which are most numerous on the limbs, are those of erythema multiforme. This is probably evidence of former streptococcal infection."

Dr. Todd reported that the antistreptolysin titre was 200 units (normal up to 50 units), which indicated some recent streptococcal infection.

Blood-counts.—9.8.34: R.B.C. 4,272,000; Hb. 49%; C.I. 0·6. 16.10.34: R.B.C. 6,020,000; Hb. 65%; C.I. 0·54. Sedimentation rate 37 mm. at one hour (normal up to 10 mm.).

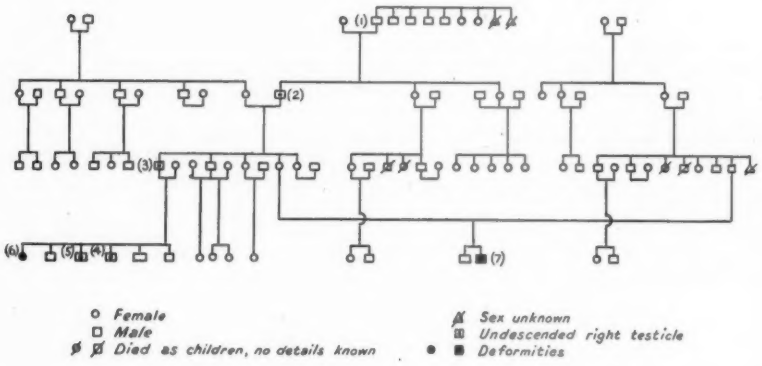
Dr. PARKES WEBER said he had seen a well-marked case of Still's disease in a girl, aged 11 years, who had a conspicuous papulo-erythematous rash. It was described in the *Trans. Med. Soc. London* (1933, lvi, 55) by Dr. H. E. A. Boldero, who said he knew of other cases with a similar eruption.

Family Tree, showing Hereditary Undescended Right Testicle and Associated Deformities.—PHILIP WILES, F.R.C.S.

This family tree is placed on record to show the transmission of an undescended right testicle through three generations, and the occurrence of grosser abnormalities in the youngest generation. It is probable that the occurrence of severer deformities, in a family already afflicted with a minor one, is not uncommon, but it is seldom possible to illustrate it so clearly.

Mr. C., the founder of the family, numbered 1 in the tree, had four brothers who all lived to over 90 years of age, but it has unfortunately proved impossible to

discover if he or his brothers had undescended testicles. In each of the affected males, Nos. 2, 3, 4 and 5, it is the right testicle that is undescended, and they have transmitted the defect to about half their male offspring. The female, No. 6, is now 22 years old, and is stated to have stunted fingers on the right hand, but it has been impossible to examine her personally. The male, No. 7, is now aged 3 years, and has only two phalanges to all fingers, except the fifth right little finger, with partial webbing, complete absence of the right foot and complete tongue tie.



The tree was entirely compiled by the mother of No. 7, and there is every reason to believe that it is accurate. She spent some six months over it and has given me details of age, cause of death or present health of the majority of the people in it.

I am indebted to Mr. E. P. Brockman, who has No. 7 as a patient at the Royal National Orthopædic Hospital, for permission for publication.

Section of Neurology

President—S. A. Kinnier Wilson, M.D.

[*October* 18, 1934]

DISCUSSION ON UNSETTLED QUESTIONS OF NEUROSYPHILIS

Dr. S. A. Kinnier Wilson (President).—Almost a century has elapsed since Donne (1837) described a spiral micro-organism found by him in the pus from primary syphilitic genital sores, and almost 30 years since Schaudinn and Hoffman (1905) likewise found a regularly spiralled body, their discovery being hailed with enthusiasm and its genuineness soon amply confirmed. It would be difficult, however, to find another instance of the fact that the research crowned by the detection of the apparently causative organism has started more problems than it has settled, or of the uncertainties that still gather round a question imagined decades ago to have been solved.

The terms *Treponema pallidum* and *Spirochæte pallida* are still used interchangeably; but since the former has a botanical, and the latter a zoological, implication the fact that both are in use merely reveals our continuing ignorance of the exact nature of the organism. Some authorities contend that it should be classified as a protozoon, others that it is one of the higher fungi, its growth taking place by budding. Others, again, adopt the view that it occupies a position between protozoa and bacteria, a sort of missing animal-vegetable link. Curious that on so fundamental a point opinions still differ after thirty years! The general claims still advanced are that it fulfils Koch's postulates; its morphology is known; it can be cultivated outside the body and kept going for long series. Yet there are good reasons for questioning more than one of them. Of the life-history of the spirochæte astonishingly little is known. Termed "leucocytozoon syphilidis" by McDonagh, that worker described its development in a complex life-cycle, from a sporozoite, through sexual (inclusive of "spirochætal") forms, to a minute infective spore. To-day, twenty years later, we can only judge it remarkable that McDonagh's researches have been largely ignored, yet, when repeated, have been to some extent confirmed. According to Levaditi and others, the types of the organism range from an inframicroscopic particle to the full-fledged spiral. Whether it is genuinely filtrable or not is uncertain, and even whether it can be cultured. Spirochætes found in culture media containing syphilitic tissue are believed by some to have only migrated from it. How it grows or divides is equally indefinite. Nothing is known of what happens to it during incubation periods, which are at times immensely long. For various reasons a pleomorphic existence seems requisite, but its details are elusive to a degree. Arsenic kills the treponeme in vitro but does not always cure syphilis by any means; old cultures and subcultures lose their infectivity, as though some intermediate phase were lacking; at an early period lymph-glands are actively virulent despite absence of the spirochæte from their tissues. Pathologically, many have been struck by the discrepancies between the size of the lesions and the number of treponemes present.

In a recent review Ingraham has collected and analysed 18 instances from the literature of both experimental human and animal syphilis in which infection has been produced by diseased tissue apparently free from recognizable spirochætes.

Whether recognizable and separable strains of treponeme exist, having a special affinity for soma and nervous system respectively, and distinguishable therefore as dermotropic and neurotropic, is a question that has been discussed at great length. On the whole, the evidence is against the dualist theory, though the matter is not perhaps finally settled. The following serious objections to it may be briefly outlined.

(1) Somatic syphilitic lesions, e.g. syphilitic aortitis, are often found in cases of general paralysis.

(2) Considering the frequency of tabes and general paralysis, conjugal examples are by contrast quite rare. The offspring of parents suffering from either may show signs of somatic infection only, as may sibs of juvenile paretics. The latter may show Hutchinsonian teeth and other somatic stigmata—all this is well recognized.

(3) When syphilis, affecting different members of a family at different times and places, is followed by the same nervous type in them all, a familial predisposition to nervous disease accounts for the fact much more simply than the conjecture that by some singular chance a neurotropic virus has been the cause of each.

(4) Arguments founded on the alleged rarity of neurosyphilis in certain countries where the constitutional variety is rampant are losing any significance they may once have possessed. Newer knowledge is proving their unreliability.

(5) Meningeal and fluid changes often occur at the time when skin and mucosæ are the seat of secondary rashes.

(6) In 81·9% of 72 cases of general paralysis (Bolton) strong evidence of psychopathic heredity was secured, discounting the need for a neurotropic hypothesis. The persons who become affected are "dementable psychopaths" and not individuals tainted haphazardly.

(7) Lowered pH of the blood, according to studies by Marinesco, forms a milieu unfavourable to treponeme growth; the higher the alkalosis of a Ringer's solution the longer does the parasite continue mobile in it. The pH of paretics (blood and fluid) is stated by him to tend to alkalosis, varying directly with the severity of the lesions, though this work needs confirmation.

(8) Recent discoveries by Kolle, Schlossberger and others seem to show that spirochætes acquire the property of neurotropism by asymptomatic sojourn in the brains of mice; when injected thereafter into rabbits they penetrate the nervous system, where they persist and multiply.

It is clear, then, that the terrain is at least as important as the germ, if not indeed more so; on receptivity, preparedness, amount of inoculum, and other factors intrinsic and extrinsic, more depends than on the quality of the virus.

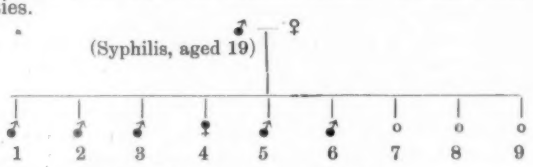
Syphilitic inheritance is another question enshrouded in obscurity.

In what form infection is transmitted from parent to offspring is unknown. Presumably it must be by the seminal fluid to the ovum that is being fertilized, yet the spirochæte is larger than the head of a spermatozoon; accordingly, we are compelled to assume that one of its forms is a minute, infective, highly resistant granule. This might simplify numerous difficulties that face the clinician, one of which concerns the occurrence of positive Wassermann reactions years after infection, and without clinical manifestations of an active—or, it may be, of any—kind.

Being of some size and actively motile, the organism could hardly enter the ovum and multiply therein without destroying it; besides, its occurrence actually within a cell seems to be extremely rare. Hence most suppose that the mother is syphilized by infected semen, the product of conception becoming diseased in turn by maternal blood viâ the placenta, or possibly during parturition. If these things are so, syphilis is never "hereditary," while the word "inherited" can mean no more than that a detachable and not ingrained condition is passed on. Yet this view encounters the objection that maternal blood may be negative and treponemes found in the tissues of the dead fœtus or infant (McCord).

The most remarkable case of the kind with which I am acquainted is that reported by Williams. A woman's sixth pregnancy resulted in biovular twins, of which one was syphilitic and died, the other being normal and surviving. She admitted intercourse with a syphilitic person as well as with her healthy husband. Her Wassermann reaction was never positive, though examined repeatedly, and she was clinically free of all syphilitic signs. No fewer than 11 subsequent pregnancies were normal. The facts surely suggest that on one occasion one ovum was fertilized by the syphilitic paramour, and that one of his spermatozoa was the vector of the spirochæte in some possibly granular form.

Sometimes the infected child is single, sometimes one among others who are healthy (rare though this is); he may be the sole survivor, or, again, he may suffer from neurosyphilis and his sibs from somatic forms. A family tree illustrates some of the possibilities.



1. Died, aged 5 years, from "inflammation of brain."
2. Premature; lived one hour.
3. Died, aged 3 months. Congenital syphilis.
4. Died, aged 16 months. Congenital syphilis.
5. Living, aged 21: juvenile tabes with optic atrophy and testicular aplasia.
6. Died, aged 12 months, from "inflammation of brain."
7. Miscarriage.
8. „
9. „

Here the whole family was syphilized, but in different ways. In another (Nonne) the record well exemplifies the medley of clinical forms that inherited syphilis may assume, being as follows, for five children:—

1. Syphilitic (serum positive, no symptoms or signs).
2. Fröhlich's syndrome (boy, serum positive).
3. Normal.
4. Juvenile tabes (girl).
5. Syphilitic (girl, congenital keratitis).

I have not been able to discover if earlier recognition and more intensive treatment have begun to affect the incidence of congenital forms; perhaps it is too soon to say. Practically nothing is known of any "laws" which syphilis may observe as it descends amid the offspring of diseased parents, with or without overt signs of its existence; we are ignorant of the reason for choice among sibs, for appearance of one clinical type rather than another, for presence or absence of somatic accompaniments. Congenital syphilis has vagaries of its own that still baffle solution.

Some of the peculiarities of adult neurosyphilis deserve note. Certain clinical varieties must be regarded as distinctly rare, though no reason for their rarity has ever been offered; indeed, in view of the diffuse character of the infection it is hard to see why they do not occur more often.

Acute ascending meningomyelitis is one of them, only a few cases being on record; in Barth and Léri's a girl of 17 developed a sudden paraplegia six months after a vulvar chancre, succeeded by rapid involvement of arms, neck, and cranial nerves, death resulting on the seventeenth day. A Landry syndrome (six years after infection), observed by Macnamara, ended in recovery, but was followed much later by tabes. In both of these cases *Micrococcus tetragenus* abounded in the spinal fluid—a very curious coincidence; its rôle being saprophytic rather than

pathogenic as a rule, it conceivably acted as a sensitizer. Whatever the explanation, the fact merits more than passing attention.

Another rare type is the systematized spinal type, consisting in the union of lateral and posterior column sclerosis with spinal amyotrophy, of which one of the best-described cases we owe to Gordon Holmes. Some years ago a similar case (but without pathological confirmation) was under my own care; a positive Wassermann reaction and Argyll-Robertson pupils were conjoined with a clean-cut ataxic paraplegia (loss of deep reflexes, extensor plantars) and a progressive muscular atrophy of hands and feet. The lightning pains of tabes were conspicuous by their absence, while in Holmes' case the dorsal roots showed no degeneration. The facts suggest that syphilis or its toxins can on occasion produce a genuinely systematized spinal disease, but the " why " of the matter eludes us.

Syphilitic Parkinsonism is rare, out of all proportion to other neurosyphilitic variants, and syphilitic amyotrophy is far from common. For some reason that escapes discovery, the latter does not seem to occur in juveniles, despite the great number of congenital syphilitic forms; at least, I do not recall ever having seen a case, nor am I acquainted with any in the literature. Oddly enough, too, these two varieties are extremely resistant to ordinary specific treatment, though why this should be so is altogether obscure. The relative immunity of the basal ganglia to syphilis is a problem seldom discussed, though some day it may prove illuminating; the intractability of the condition if it does arise distinguishes it also from most of the other kinds.

As regards optic atrophy, the extreme rarity of central scotoma is singular, not to say inexplicable. Pathologically, interstitial lesions round the nerve probably account for the common clinical type, that which is characterized by loss of peripheral vision; patchy processes may account for patchy fields; parenchymatous lesions may perhaps entail the other clinical type, distinguished by diffuse impairment of visual acuity, with dyschromatopsia. If toxic invasion be part of the disease I am at a loss to explain the absence of attack on visual fibres that succumb so often in other circumstances.

The question of the relation of symptoms to lesions, and of the pathogenesis of the former, bristles with difficulties so far as numerous aspects of neurosyphilis are concerned. It is a sound interpretative rule to follow Hughlings Jackson's dictum that positive symptoms cannot be caused by negative lesions, and to apply this to the diversified symptomatology of the disease. A tabetic does not walk badly with his posterior columns, but with what of his spinal cord is left. The doctrine is of material value in any analysis of neurosyphilitic manifestations, whatever their nature. It shows that many of the clinical phenomena of general paralysis, for example, are release phenomena, due to the unimpeded activity of mechanisms that are normally more under control. In this way we can account for the megalomania of some 50 per cent. of cases, more or less; what it does not explain is the absence of this symptom in the remainder. Does this absence depend on the personality that is breaking-up under the ravages of the disease, and not on the latter directly? If that be the case, the symptom should occur far more often when other infective processes assail the cerebrum: yet it does not. Difficulties also surround the question of insight in the same form of neurosyphilis; preservation of insight is curiously unequal and variable. I am not likely to forget the impression left on me by seeing a well-known histopathologist in the grip of the affection; he never knew that he was suffering from the morbid state whose pathology he had himself minutely described. By way of contrast I may cite the case of Guy de Maupassant, who died a paralytic, and who nevertheless was able to utilize his own experience of " autoscopy " in his graphic story " Le Horla."

The pathogenesis of such tabetic phenomena as visceral crises and trophic lesions needs much further investigation. As regards the latter, different theories

hold the field. The problem depends for its solution in some measure on ascertaining which of the lesions are primary and which secondary. Since trophic change occurs in other syphilitic forms than the tabetic, not seldom appears early or in "fruste" varieties, and is seen also in syringomyelia where vascular disease does not constitute a factor, its basis is more probably neural; but whether this consists of damage to deep nerves supplying the tissues directly, or to neurovascular filaments, or possibly to the spinal representatives of either, has not been discovered. Even greater obscurity involves the question of visceral crises. What is most difficult to understand is the cumulative and explosive character of the phenomena, and their periodicity. These features naturally enough suggest a relation to epileptic discharges, less brief and longer drawn out than usual. Byrnes' speculation is to the effect that "banking of potential" occurs in sympathetic neurones distal to the lesion as a consequence of the latter, and that "energy" accumulates to such a degree that the block can no longer prevent the passage of impulses to cord and brain at the same time as discharge begins locally.

I can only allude in passing to the extremely troublesome matter of the interrelation of clinical symptoms and laboratory findings, though many instances crowd the mind. Other participants in this discussion will deal with the problems it arouses.

Finally, something must be said of advances in the treatment of neurosyphilis and of their effect since they came into vogue. I must however content myself here with reference to one point chiefly, viz., the evidence of mortality tables in respect of general paralysis and of tabes.

ACTUAL NUMBER OF DEATHS FROM GENERAL PARALYSIS (MALE AND FEMALE) FOR ENGLAND AND WALES, IN BIENNIAL PERIODS

Years	Males	Females
1913-14	3,563	842
1915-16	3,558	805
1917-18	3,671	767
1919-20	2,606	506
1921-22	2,652	583
1923-24	2,659	642
1925-26	2,415	562
1927-28	2,379	547
1929-30	1,931	482
1931-32	1,809	495

ACTUAL NUMBER OF DEATHS FROM TABES DORSALIS (MALE AND FEMALE) FOR ENGLAND AND WALES, IN BIENNIAL PERIODS

Years	Males	Females
1913	615	117
1913-14	1,215	237
1915-16	1,310	211
1917-18	1,223	219
1919-20	1,103	169
1921-22	1,241	252
1923-24	1,224	249
1925-26	1,232	246
1927-28	1,293	264
1929-30	1,323	260
1931-32	1,191	258
1932	634	144

From these figures we can see at once that the mortality of general paralysis has been almost halved in the course of the last twenty years, whereas that of tabes has not, to all appearance, been modified in the least. I confess there is here provided a contrast the explanation of which is far from clear. Since the salvarsan era the two major varieties of neurosyphilis have been submitted to identical treatment, more or less; one has responded and the other has not. I do not think that the introduction of arsenicals and of malaria, etc., is alone responsible for reducing the death-rate of

general paralysis, except in so far as they have meant superior treatment for the developed disease—at all events, figures adduced by Smith seem to indicate that the modern treatment of the primary disorder has not been of itself instrumental in diminishing the incidence of paretic sequelæ. Perhaps the explanation lies in the fact that statistics even from pre-salvarsan times show how little tabes tends to shorten life, whereas general paralysis is an acute or subacute malady of a more serious kind.

Dr. J. E. R. McDonagh: I do not think I can do better than to epitomize the inferences I have drawn from my clinical and research work on this subject. In my opinion all matters relating to syphilis must remain unsettled until the knowledge we think we have acquired since 1905 is resifted, and the behaviour of the infection in the body is looked upon as being no different, except in degree, from any other invasion, be it microbic, physical or chemical. I feel sure that a re-examination of the three main tenets would reveal: (1) That the spore of a coccidial protozoon is the actual cause of the infection—the *Spirochæta pallida* being no more than the adult-male phase in the life-cycle. (2) That the serological tests are crude and non-specific, and cannot be used as indicators of either the presence or the absence of the infective phase. (3) That treatment, equally non-specific, does no more than correct the abnormal chemico-physical changes to which the protein particles in the plasma, the host's main resistance, have been subjected by the parasite.

The acceptance of a protozoon having a complicated life-cycle, which has to be completed before signs and symptoms manifest themselves, explains the long incubation period, conceptional and congenital syphilis, and why the word "cure" should never be used. The spore may exist in perfect harmony with the cells of its host for an indefinite period, and awake to go through its life-cycle when the patient's resistance becomes lowered. In the quiescent state the spore cannot be detected, but its presence in the body appears to be lifelong, because a patient who has once had syphilis never becomes reinfected. The whole behaviour of the micro-organism in the body is determined by the state in which the host's resistance happens to be at the time of infection, and throughout the infection. It is the body, not the parasite, which varies, hence it is incorrect to speak of a neurotropic and dermatropic type of micro-organism. Any factor which alters the patient's resistance may change the course of the infection. The years the infection has been extant may be one of the factors, and treatment is another. Injudicious treatment has the action of an invader, and there is a definite relationship between the length of time the infection has prevailed in a community and treatment, and the incidence of involvement of the central nervous system. Syphilis, changing the blood in the way it does, is particularly liable to precipitate manifestations of disease, the result of arteriosclerosis. The infection causes premature senility, and aggravates other invasions which have a similar effect. Senile dementia, Parkinsonism, chronic anterior poliomyelitis, amyotrophic lateral sclerosis, certain cases of posterior-column degeneration, etc., rest upon a basis of arteriosclerosis, which has been precipitated by syphilis but not caused by it. It is necessary to make this distinction because antisyphilitic treatment tends to aggravate arteriosclerosis, and in the conditions mentioned it frequently does more harm than good. The basis being arteriosclerosis explains why the conditions are not met with in congenital syphilis. The trophic lesions tend to fall into the same category, but blood-changes play an important rôle in their production, as evidenced by the fact that if treated early they respond extraordinarily well to insulin.

I have known Charcot's arthritis, spontaneous fractures, perforating ulcers, etc., mend with ten injections of this remedy. Experience has taught me that infinitely more is to be gained by treating the patient than by treating his infection. And I am

convinced that if syphilis and all other invaders were removed from their pigeon-holes, collected together and regarded as the single cause of extrinsic disease an enormous advance would be made. The only reason why syphilis and no other infection causes congenital disease is because it occasions the chemico-physical change in the blood which links inherited disease to the extrinsic form. Even when we have got as far as linking the forms of disease together we are no more than on the threshold of our journey. Disease is a natural sequence of life and to close my contribution to this discussion I would say that syphilis behaves in augmenting disease in the same way as any other invader. In short there is nothing in syphilis *qua* syphilis.

Dr. B. Buckley Sharp: I propose to confine my remarks to the problem of treatment. To the clinician, the crux of the problem is how to bring therapeutic substances into contact with the parenchymatous tissue of the central nervous system in which the infecting organism lies embedded—in other words, how to penetrate what is termed "the blood-brain barrier."[1]

There is no known anti-spirochætal serum and no vaccine available for therapeutic use. Recourse has therefore to be made to chemotherapy aided by the ability of the patient to defend himself against infection by his own immunity response and by his ability to react appropriately to the chemical substance injected into his blood, muscles or subcutaneous tissues.

Chemotherapy is not just a simple process involving direct action of the drug upon the organism, like the action of an antiseptic. Some other substance is formed by interaction between the injected drug and the patient's tissues which has not been identified but which is thought to be a protein and to have a molecule too large to pass through the intact capillary wall. The same circumstances are thought to apply whether we are considering the arsenobenzol group of drugs or bismuth and its various salts. I shall not here refer in detail to other metals that are found to have a spirochæticidal action in experimental syphilis, namely, vanadium, antimony, tellurium, platinum, gold, mercury, indium and gallium.

The first general question I wish to raise concerns the patient's own immunity response and is important in considering the prophylaxis of neurosyphilis. Should intensive treatment be instituted at the earliest possible stage of the initial infection in the hope of stamping it out by a chemotherapeutic mass attack? Or should time be allowed to elapse for the development of the patient's own immunity response before active therapy is started? In practice it is usual to start treatment immediately syphilis is diagnosed, and complete cure is quite commonly effected by this means in sero-negative primary syphilis as judged by subsequent primary reinfection. On the other hand it is unusual for a patient who develops a vigorous reaction in the skin in the primary and secondary stages to develop neurosyphilis subsequently. I believe that neurosyphilis is most likely to follow in cases that have been inadequately treated at the start (sufficiently to inhibit a good immunity response but insufficiently to eradicate the disease), and in cases in which the early manifestations of the disease are so slight as to pass unrecognized owing to the failure of the natural reaction to infection.

My second general question is—what drugs can most readily penetrate into the nervous system and how can such penetration be promoted? To gain its objective the therapeutic substance must pass through the choroid plexus or traverse the perivascular spaces in the brain and spinal cord. Thus, in theory, any substance that can so pass should be recoverable from the cerebrospinal fluid unless it is altered beyond recognition.

Arsenic.—The trivalent arsenobenzol compounds in general use do not normally pass the blood-brain barrier. In 1923 Voegtlin, Smith, Dyer and Thompson found that the pentavalent compound tryparsamide possessed a higher penetrability for the

[1] Friedmann and Elkeles, *Lancet*, 1934.

central nervous system than did arsphenamine or neoarsphenamine, and subsequent therapeutic use of this drug in neurosyphilis, particularly in G.P.I., proved this to be the case. It appears also to have a special affinity for the cells of the central nervous system, which accounts for its toxic effect upon the nerve-cells of the retina in some cases, causing amblyopia with contraction of the fields of vision, going on to blindness if its exhibition is not stopped in time. If this effect is produced it will occur early and after from 10 to 15 injections it need not be feared. Lees, from his experience and from the literature, put the incidence of temporary amblyopia at 3-10% and of permanent damage to vision at 0·5-3%.

This drug is of great value in general paralysis and may produce complete and lasting remission in early cases, of which the following is a good example: A professional man aged 62 developed general paralysis while under treatment with a trivalent arsenobenzol drug for aortic regurgitation of syphilitic origin. The exhibition of tryparsamide caused a rapid clinical recovery which has remained complete for over five years and he was soon able to resume his professional work and is still in active practice. Henderson and Fong (1928) claim that in early cases of neurosyphilis results of tryparsamide treatment are about the same as those of malaria. Tryparsamide has little if any effect upon the somatic, as opposed to the neurological, manifestations of syphilis and therefore should be combined with bismuth therapy. There are several other effective pentavalent compounds such as stovarsol and acetylarsan—to mention only two of them.

Bismuth (insoluble, lipo-soluble or water-soluble) is only recovered in minute quantities from the brain and cerebrospinal fluid after intramuscular injection and no readily permeable form has been found, although American workers affirm, and Levaditi denies that "iodobismitol," a soluble compound of sodium iodobismuthite and iodine, has this property. (*Bull. de la Soc. Franc. de Derm. et Syph.*, 1933, xl, No. 5, 738.)

The electrical charge carried by the metallic ion is said to be of importance in determining its penetration into the central nervous system (Hanzlik and Spaulding, 1931). Therefore in some bismuth preparations, such as iodobismitol and sodium bismuthate, the bismuth ion is negatively charged and, in theory at least, of greater penetrating power than the positively charged bismuth ion present in bismuth salicylate, which is the official pharmacopœial preparation. This is the opposite of the behaviour of toxins, of which Friedmann and Elkeles state that neutral and positively charged toxins pass the blood-brain barrier while negatively charged toxins do not.

Are there any artificial means by which penetration of the central nervous system can be promoted? It is well known that inflammation has this effect, and if it were possible safely to produce an aseptic meningitis something might be achieved in this direction. Injections of air into the subarachnoid space have been used with this end in view. I would suggest tentatively that intrathecal or intracisternal injections of horse serum might be of value. Spinal drainage following intravenous injection of one of the arsenobenzols has been performed in the hope that some of the drug may thereby be persuaded to leak through the choroid plexus. Hypotonic solutions might be injected intravenously along with the drug, with the same idea. But the success of these manœuvres seems unlikely.

Artificial pyrexia.—Sträusler, Koskinas and Greenfield have found that the brains of general paralytics dying during malarial treatment show an unusual increase in the inflammatory and proliferative processes, whereas in those who die later the inflammatory signs have retrogressed. This suggests that in pyrotherapy we have a means of assisting the penetration of drugs into the nervous system, and is an argument in favour of giving them along with the fever treatment and continuing them after it. I am well aware that lasting remissions in G.P.I. occur after malaria alone. But, for my part, I do not think we are justified in withholding follow-up treatment, on and off, for an indefinite period.

All three types of malaria have been used in pyrexial treatment, and no doubt Dr. Nicol will tell us about them. There are many methods besides malaria of inducing bouts of pyrexia : intravenous T.A.B., intramuscular sulphur injections, injections of pyrifer, diathermy, Wilde's thermal couch, and infection with relapsing fever. There is no doubt that malaria obtains the best results, but why this is so is not clear. One can only postulate some specific property in addition to the production of fever with its accompanying leucocytosis and its increase in metabolism. This raises questions as to whether naturally acquired malaria can prevent or reduce the incidence of neurosyphilis, and whether symptomless patients with persistent changes in the cerebrospinal fluid despite treatment on the usual lines should be submitted to malarial infection, a treatment which is decidedly unpleasant and not entirely devoid of risk to life. On the first of these two points I have been able to collect evidence that neurosyphilis occurs in Europeans who are constantly suffering from malarial attacks in the tropics, but the coloured races in malarial countries are rarely affected. The American negro and the Chinese are not racially immune.

Time does not permit me to submit the evidence in detail.

Results of malaria-therapy in children with congenital neurosyphilis are very disappointing, and will no doubt be dealt with by Dr. Nabarro.

Are intracisternal or intrathecal injections of salvarsanized serum of value in general paralysis and tabes respectively? This method has been criticized on the theoretical grounds that there is no evidence that any therapeutically active substance is being administered in the serum withdrawn 1 to 2 hours after intravenous injections of arsenobenzol; that the amount of therapeutically active substance, if any, must be infinitesimal in 40 c.c. of blood withdrawn, and that the material injected does not reach the site of the infection in the brain. It is stated by Harrison that after intravenous injections of 606 and 914 these substances circulate as such for no more than two hours, during which time the blood-serum shows a definite anti-spirochætal power. Therefore one hour after injection when the blood is withdrawn there must be some unchanged arsenobenzol and some of its anti-spirochætal end-product present. As regards the amount that may be present in 40 c.c. of blood, this need not be so infinitesimal as is suggested in relation to the size of the brain, when we consider that 40 c.c. of blood is about 1/150 of the total blood volume, and the weight of the brain is about 1/45 of the total body-weight. Substances injected into the cisterna magna pass over the surface of the brain to drain away in the venous sinuses, and also can diffuse into the ventricles; whether they can pass from the subarachnoid space into the substance of the brain is more problematical. Another theory of the beneficial action of this method is that salvarsanized serum contains the product of lysis of spirochætes, thus setting up a local immunity response.

Whatever may be the theoretical pros and cons, a small series of cases put forward before a joint meeting of the Sections of Neurology and Psychiatry, by Purves Stewart in February 1929[1] suggested that in general paralysis the results of malarial treatment followed by a course of intracisternal salvarsanized serum gave a higher remission rate than malarial treatment alone, and that the return of the cerebrospinal fluid to normality was more frequent. I have not seen much reference recently to the use of this method.

From the clinical point of view, I find the most perplexing and disheartening problem to be that of tabes in its early stages, particularly in respect of lightning pains and crises, and of optic atrophy. Tabes in general may continue to progress despite treatment, or may remain in a stationary condition for years without treatment. It is difficult to assess in any given case how much good, if any, we are doing by treatment.

[1] *Proceedings*, 1929, xxii, 842.

I know of no treatment along medical lines that will with certainty put an end to root pains and crises. Prolonged and intensive treatment with silver-salvarsan, iodides, bismuth, tryparsamide or mercurial inunction cannot be relied upon to do so. I have a patient now whom I have been treating on and off for eleven years on these lines. But from time to time he has very acute attacks of lightning pains, although in this period loss of function has been negligible. I have tried malaria in two cases, with only temporary benefit as regards the pains. X-ray treatment of the spine has been advocated, but this again is probably only of temporary benefit.

Optic atrophy seems to go on to blindness eventually, whatever one may do. Various special lines of treatment have been suggested, including malaria, endolumbar salvarsan injections, and intracisternal injections of mercuric chloride. Of these methods I have no personal experience. But, fortified by the writings of the late David Lees, I have ventured on tryparsamide injections, despite the known toxic effect of the drug on the optic nerve in some cases. My experience is, however, too limited to draw conclusions, but the results obtained and published by Lees give some hope for this method.

Dr. David Nabarro: I shall confine my remarks almost entirely to congenital neurosyphilis, to which I have devoted particular attention. It is a big subject, so that in the time at my disposal I shall draw attention to some of its more important points. An excellent survey of congenital neurosyphilis is given by Dr. Ferguson and Dr. Macdonald Critchley in the *British Journal of Children's Diseases*, 1929, 1930, xxvi, xxvii, but apart from that paper little has been written upon the subject by British authorities. Various continental and American authors, among them Bresler, Ravaut, Gaucher, Jeans, and Kingery, have written papers upon the subject. The largest reported series is that of Jeans and Cooke, 645 cases, an account of which will be found in their book. One curious fact emerges from their figures, namely that whereas one-twelfth, or 8%, of syphilitic white infants up to 2 years of age have *active* neurosyphilis, and two-fifths, or 40%, have neurosyphilis (abnormal cerebrospinal fluids), in the case of coloured children the figures are respectively one-twentyfifth, or 4%, and one-fifth, or 20%—just half. In older children the difference is still more marked; one-sixth, or 16·7%, of all older syphilitic white children have serious neurological lesions, whereas only one-sixtyfourth, or 1·6%, of coloured older children are similarly affected. The numbers investigated were 249 white and 120 coloured children over 2 years of age. It is difficult to explain why white children were ten times as susceptible as coloured children, unless it is due to a racial immunity or results from native malaria. Coming to our observations at the Hospital for Sick Children, Great Ormond Street, I have now examined the cerebrospinal fluid of more than 500 children with congenital syphilis, and a short time ago I analysed the results we had obtained in our then total of 428 cases. Of these 428, 107 were aged under 1 year, 321 over 1 year; of the 107 infants no less than 57 (53·3%) had abnormal spinal fluids, and of the 321 older children 63 (19·6%) had abnormal spinal fluids; but of these older children many were not examined until late in the treatment, owing to the fact that we had no ward accommodation for these cases until 1923, and doubtless many children had positive spinal fluids originally which had responded to the treatment given. If these cases be deducted from the total 321, then there remain 130 cases of children over 1 year of age whose spinal fluids were examined early in the treatment, with 38 = 29·2% positive.

These figures show how frequently the central nervous system is invaded in congenital syphilis, yet how often is a lumbar puncture done in these cases unless or until a catastrophe such as the onset of a hemiplegia, a fit or other symptom of meningo-encephalitis, draws attention to the involvement of the central nervous system, by which time irreparable damage has been done? As is the case in adults,

so I believe it is in infants, that insufficient early treatment, particularly with mercury, may lead to neuro-recurrence in the second or third year or possibly later, if the nervous system is invaded at birth or shortly after. For this reason I advocate a routine lumbar puncture in all cases of congenital syphilis, and when the fluid is found positive, I impress upon the mother the importance of bringing the child regularly for treatment. In my experience, the cerebrospinal fluid, when positive in young infants, practically always becomes negative as a result of regular and thorough treatment either with injections of arsenic and oral mercury or with injections of bismuth alone. In only three cases have I seen a spinal fluid become strongly positive after having been negative, but in two of these it subsequently became negative; the third has only just been discovered and is about to be treated.

Neurosyphilis in children past the stage of infancy may be latent or manifest, the former being diagnosable only by carrying out routine lumbar punctures in all cases of congenital syphilis. The symptoms of congenital neurosyphilis have been fully described in the paper by Ferguson and Critchley referred to above. In my experience a mild degree of hydrocephalus is not uncommon in these cases; some patients may have unequal and sluggish, or fixed, pupils as a sign—sometimes the only sign—of neurosyphilis. Disorders of conduct or behaviour, fits of temper, moral obliquity, varying degrees of feeble-mindedness, and occasionally mental precocity may be met with in these cases. When I was pathologist at the West Riding Asylum, Wakefield, about twenty-five years ago, we had a small number—I think about six cases—of juvenile G.P.I. in the wards, but these were all fairly advanced and obvious cases clinically and serologically. Arsenical treatment was not being given at that time. Since I have been at the Great Ormond Street Hospital I have found it difficult to diagnose G.P.I. in early childhood because of the usual absence of facial immobility and the psychological changes commonly noted in adults. In children with progressive mental deterioration and occasional fits, during which the cell count in the cerebrospinal fluid may be greatly increased, one has been in the habit of regarding the case as one of G.P.I. if the Lange gold curve was typically paretic. In my experience cases of juvenile tabes are extremely rare. I can recall only two during my twenty years at the Great Ormond Street Hospital in which the most important symptom was defective vision due to optic atrophy. No marked sensory or motor symptoms supervened, and one of the patients did very well on intravenous sodium iodide treatment in association with arsenic, the blood and cerebrospinal fluid becoming negative. The other patient, a girl, in spite of treatment by sodium iodide, malaria, bismuth, and arsenic did not show any improvement after four years' treatment, the blood and cerebrospinal fluid remaining strongly positive. The boy started his symptoms at about 8 years and the girl at about 9 years of age.

Neurotropic v. dermotropic spirochætes.—We have examined two or more children in a family in the case of sixty-one families with the following results: In 39 families both children gave negative results; in 16 one gave positive and one negative; and in six families two or three children were positive. No definite conclusions can be drawn, however, from these figures, for even if six out of 61 families with neurosyphilis in two or more of the children be considered unduly high, it is impossible to say if this indicates a neurotropic strain of spirochæte or a particular susceptibility of the nervous tissues in these families.

For some years I have been interested in the question of third-generation syphilis, and last year I made it the subject of my presidential address before the Medical Society for the Study of Venereal Disease. Details of thirty-one families were given in which syphilis was *undoubtedly* or *probably* present in three generations, and in nearly every one of these cases the disease, I believe, was transmitted to the third generation by an untreated congenitally syphilitic mother, though of course I

admit that scientific proof is lacking, inasmuch as it is impossible to be certain in the majority of cases that the father may not also have had syphilis. I have made two interesting observations in this connexion: (1) That it is frequently the second child which is most markedly affected, the first child being either healthy or only slightly affected; (2) that in several of the cases the affected child has shown an involvement of the central nervous system. It is a curious fact that of the three children I referred to previously in whom a positive cerebrospinal fluid was found after it had been negative at an earlier examination, two were cases of third-generation syphilis. Sometimes the child of a congenitally syphilitic mother is mentally deficient or has epilepsy, without the child showing a positive Wassermann reaction in its blood or cerebrospinal fluid.

Further observations on these points are desirable in order to ascertain if these were isolated and accidental happenings, or if they are usual occurrences.

Coming now to the question of treatment, I will endeavour to deal with this aspect of the problem as briefly as possible. If an infant under 1 year shows involvement of the central nervous system, the condition practically always clears up if a thorough course of treatment is given—either arsenic and mercury by mouth or even injections of bismuth alone. At the end of a year's, or at the most two years' treatment, in most cases the blood and cerebrospinal fluid become negative, and hitherto have remained so. In the case of older children if the neurosyphilis is latent, or perhaps unequal pupils are the only signs of the condition, one gives thorough treatment for two years with periodical examinations of the cerebrospinal fluid and the blood. If the fluid is not negative at the end of this time, injections of tryparsamide are given. If the condition does not improve malaria may then be tried. In the pre-tryparsamide days I treated several children who had neuro-syphilis with intracisternal injections of salvarsanized serum and with malaria, and it appeared to me that rather better results were obtained by using both forms of treatment than by using either one alone. In a very large number of cases I have succeeded in making the blood and spinal fluid negative, but this does not necessarily mean that the patient is cured. We must remember that in most of these children considerable damage has already been done to the developing central nervous system, and although the infection may be eradicated, the pathological effects upon the brain and the impairment of its function will persist. I have had a striking instance of this in the case of a child about 6 years old, who appeared to be smitten with an attack of G.P.I. She was attending school at the time, and according to the parents the onset was most abrupt, with an acute maniacal attack. On inquiry, however, from the schoolmistress, it was ascertained that the child had been peculiar in her behaviour for some months prior to the sudden outbreak. I have reported this case in the *Lancet*, 1927 (ii), so that I will not spend a great deal of time upon it, but briefly the history is as follows: The Wassermann reactions of the blood and cerebrospinal fluid were strongly positive, and the fluid gave the typical reactions of G.P.I. The child was treated for nearly two years with arseno-benzene injections, mercury by mouth, bismostab injections, malaria and two courses of intracisternal injections. The final outcome was a perfectly normal blood and fluid—the child appeared physically quite well, and the fluid and blood have remained negative for several years. When last seen, now just two years ago, she was a well-developed girl of 13, but mentally backward and almost unteachable. This case is to me very instructive, because apparently one saw it quite early, and one hoped that by energetic treatment one would be able to cure the patient. Apparently one has cured the syphilis but not the patient, and one is tempted to ask "was it all worth while?"

And finally, may I say that I have come to the conclusion, from my experience, that the two forms of neurosyphilis worth treating are that which occurs in early infancy, and the latent neurosyphilis of older children. Clinical neurosyphilis,

although it may be considerably benefited by treatment, does not, in my experience, result in the production of cure or of an individual who is going to be an asset to the community.

Dr. W. D. Nicol: I will confine my remarks to a few of the many unsettled questions which arise in the practice of malaria-therapy. At Horton, where a special centre for treatment was established in 1925, a comparatively large population of treated cases has been amassed. At the moment there are some 200 cases (110 women, 90 men). One can at once deduct from this total the admissions during the last seven months, the reason for this being that from past experience the period of from seven to nine months is almost a constant factor in assessing the value of malaria-therapy. Nearly all the patients well enough to be discharged on certificate, to their homes and the outside world, are ready for discharge from about seven to nine months following a course of malaria. In fact if a case is not sufficiently recovered to be allowed out by this time, one can say with a fair degree of confidence that that patient never will improve sufficiently. This being the case, our population of 200 cases is reduced to 84 women and 53 men (the present excess of female patients suffering from G.P.I. over males is due to the fact that from 1925 to 1931 only female cases were treated ; therapy for male cases was commenced at the end of 1931). This residue represents cases which have been treated as far back as 1925 until quite recent times at the beginning of this year. On further analysis it is found that a small proportion (12 women, 17 men) have shown marked improvement, and though not sufficiently recovered to return to the outside world, are useful workers in the hospital. In a larger proportion (28 women, 14 men) the progressive course of G.P.I. has become arrested and many of these are able to do some simple routine ward work. Finally there are those patients who are deteriorating after some years of improvement, and others who go downhill fairly rapidly in spite of treatment (44 women, 22 men). Whatever criticism may be levelled against malaria-therapy, it indisputably prolongs the life of the patient, the physical health improves in nearly all cases, and it enables many patients to return to their vocations outside. Of the residue who are not well enough to be considered recoveries, a happier and cleaner existence awaits them. Even in the final deteriorating stages one never witnesses that contracted, emaciated, semi-existence which was observed before the days of malaria-therapy.

One of the first problems which arose in malaria-therapy and remains still unanswered was: why is it that some cases respond to treatment and others do not? Many failures can be explained by the fact that the cases which did not were too far advanced, and irreparable damage to the cerebral cortex had resulted, but cases do occur in which the prognosis appears favourable, the onset is recent, and yet malaria-therapy has little if any effect. At first it was hoped that a second course of malaria-therapy would benefit the patient. At Horton it has been possible to ring the changes with different species of malaria—benign tertian, quartan, malignant tertian and *Plasmodium ovale*—in addition to giving tryparsamide or other arsenicals, but we are forced to the conclusion that if a case does not respond favourably to a first course of malaria, it will not respond at all.

Another feature of interest in this residue of failures to respond to malaria therapy, is the alteration in the character of the psychosis. In those in whom the disease is arrested or in whom deterioration is commencing, dementia is prominent. A grandiose or manic type of G.P.I. becomes a simple dementing form. Rarely will grandiose delusions persist and still more rarely (I have seen two cases) a picture of grandiosity will supervene on a simple dementing form, which hitherto had never displayed any delusions. A fair proportion of cases will develop into a schizoid type and unless one knew the previous history, these would be taken for chronic cases of dementia præcox. Then there is that very small group in which

an acute hallucinatory psychosis supervenes. At the moment I have four examples in each sex. It is described by continental workers as occurring in about 10% of cases and as being of no serious import, but in my experience it is of extremely bad prognostic significance, in some cases terminating fatally within a few months, but in others persisting for some years. It rarely clears up.

Lastly, a few words about the discharged cases of G.P.I. Malaria-therapy was introduced into this country in 1922. I have personal knowledge of some patients who have remained out eight years and are still well and at work. Dr. Golla now has a follow-up clinic at the Maudsley Hospital for discharged cases treated at the L.C.C. mental hospitals. A point of interest is that those patients who had markedly slurred speech before treatment, though improved out of all recognition, still show traces of dysarthria. Again, has an Argyll-Robertson pupil ever been observed to react after treatment? I have never seen one. As a class these patients are more grateful than any other type of mental patient: they are pleased to see one again and keen to return to hospital and see old friends. Their geniality and friendliness may be compared with those of the alcoholic. Has a diminished sense of hypercriticism introduced them to a happier and more congenial world? Many of these patients are doing skilled and useful work outside. One asks the question: How long are they going to remain well? I have had some patients relapse and return after an interval of three and four years, only to deteriorate gradually. My impression is that mental relapses in malaria-treated cases are less common than in cases treated by sulfosin and other artificial heat-producing agents. Time alone will tell us the ultimate fate of the successfully treated general paralytics.

Dr. Kenneth C. L. Paddle: I have been particularly interested in what Dr. Nabarro said on the subject of congenital syphilis. As my work is amongst mental defectives, my experience of neurosyphilis is necessarily limited to such cases. The relation of syphilis to mental deficiency is one which is even now not quite settled. In mental defectives suffering from congenital syphilis, are we to infer that the mental deficiency is the result of syphilis in all cases, or is it merely coincidence? Still states that "7% of congenital syphilitic children are mentally defective—a proportion twenty times greater than in the general population."

A careful survey of mental defectives at Caterham Mental Hospital, which included the examination of both blood and cerebrospinal fluid of 2,000 patients, showed that the incidence of congenital syphilis was 9·2% amongst children, and 4·9% amongst adults. I do not know what the incidence of congenital syphilis would be if a comparable group of normal people in this country were to be examined in the same way, but it surely must be lower. Lawrence, in the U.S.A., puts it at 2·3%.

Not all mental defectives with congenital syphilis have abnormal cerebrospinal fluids. Amongst 117 congenital syphilitics of our series, in 28, or 23·9%, the cerebrospinal fluid was abnormal. Of these 28: in 16 the Wassermann reaction was positive, in 18 there was excess of cells, in 23 excess of protein, and 20 gave a colloidal gold reaction, mainly of the paretic type. There was only one case of juvenile general paralysis, the other 27 being regarded as examples of meningovascular syphilis. 85 others gave positive Wassermann or Meinicke reactions in the blood-serum only, but had normal cerebrospinal fluids, and 4 were serologically negative. It is especially in this large group that doubt exists as to whether the congenital syphilis is related to the mental defect. Would such children still have been mentally defective in the absence of syphilis in the parents? I have obtained, by personal interview, the record of the family history of 47 unrelated cases of mental deficiency complicated by congenital syphilis, and find that in 24, or 51%, there was a definite neuropathic taint; so that in these cases, at least, it is probable

that the mental defect was the result of two factors acting together, either of which may have been insufficient, in itself, to produce the condition in question. In fact, in the absence of some other factor such as, what I may call, "an inherent neuronic weakness," I very much doubt whether congenital syphilis can be held responsible, except in rare cases, for producing mental deficiency. If this were not so, I submit that we should find a higher rate of congenital syphilis amongst mental defectives, and a much higher incidence of mental deficiency amongst congenital syphilitics.

Finally, I would like to say a word on the difficulty of assessing the value of any form of antisyphilitic treatment in congenital syphilis. I have notes on eight patients between the ages of 7 and 27, whose blood-serum gave positive Wassermann or Meinicke reactions some three years ago with, in one case, an abnormal cerebrospinal fluid, yet without any treatment whatever; all these cases now have normal blood and cerebrospinal fluid. Had they been included in any scheme of treatment, false conclusions might easily have been arrived at.

References.—LAWRENCE, J. S., "Congenital syphilis in institutional children," *Journ. Amer. Med. Assoc.*, 1922, p. 566. STILL, G. F., "The preventive aspects of medicine." Post-Graduate Series, 1934 vol. 2, p. 50, *Lancet*.

Dr. L. C. Cook: I am bringing to your attention a number of cases of congenital neurosyphilis which I have come across at the Fountain Hospital for mental defectives; to me, both their classification and their treatment are unsettled questions.

For purposes of discussion I am dividing them quite arbitrarily into two groups according to their serological findings. The first group consists of eight cases, whose serum Wassermann reaction and Meinicke-Klarung reaction are positive and whose cerebrospinal fluid shows a positive Wassermann reaction, an excess of cells and protein and a Lange curve of the paretic type. The second group is made up of nine cases showing a positive serum Wassermann reaction and Meinicke-Klarung reaction, but normal cerebrospinal fluid. I have discarded any case whose syphilis might possibly have been acquired. In the first group are to be met clinical syndromes diverging in many respects and having only three common points: (1) a cerebrospinal fluid of the paretic type, (2) some degree of mental deficiency and (3) complete absence of the clinical syndrome usually associated with congenital G.P.I. or tabes. The following is a brief summary of these cases.

Case I.—The patient is a girl now aged 18 years and 9 months, whose mental age is just over seven years. She is extremely simple and childish, garrulous, excitable and easily upset. She shows marked emotional facility, but is usually cheerful and energetic and is always immensely interested in everything around her. She is small, slight, and of degenerate appearance, but there are no definite syphilitic stigmata. She shows a marked convergent strabismus, coarse lateral nystagmoid movements, unequal, irregular and sluggish reacting pupils, lens opacities, choroiditis and some slight degree of optic atrophy. No dysarthria is present and she is not subject to fits. Admitted at the age of 8½ years—a delicate, fretful, helpless child who could only say a few words—she has steadily improved, has had excellent general health for some years past and now does useful work in the hospital laundry.

Case II.—A woman aged 25, who was admitted to hospital at the age of 14 years and three months. Her mother admits having had eleven miscarriages. The patient has a mild degree of residual spasticity of the legs and right arm, divergent strabismus, immobile pupils, choroiditis and some optic atrophy. No fits or dysarthria. She has improved to some extent since admission and now works satisfactorily in a routine manner.

Case III.—A boy, aged 9 years and 10 months whose mental age is about 5½. He shows some weakness of the left side of his face, left external squint, lens opacities, choroiditis, and unequal, fixed pupils. There is a chronic sore at the inner angle of the right eye. No fits or dysarthria. He has improved slightly since admission two years ago.

Case IV.—A boy, aged 11 years and three months, whose central nervous system does not show any abnormal signs. On admission six years ago he could not walk and his speech was limited to a few words. He has shown general improvement, walks by himself, although a little unsteadily, and talks freely in the lalling and indistinct manner common to many aments.

All these four patients are active and show a tendency to improvement.

Case V is of considerable interest and of a very different type. It is that of a girl aged 12 who was backward from infancy, but began to talk at the age of 18 months, and walked at that of 2 years. She learned to read a little, and went to an ordinary school until she was 9½. According to her mother she began to walk on her toes and "fall over nothing" at about the age of 7, and from that time deterioration, both physical and mental, set in. At 8½, however, her I.Q. was 79, and her school report showed her to be able to join in games. She is also reported to have been capable of taking messages, shopping and going alone by bus. In September 1933 she contracted scarlet fever, and two days after her return from hospital suddenly became incoherent and later inarticulate, drowsy, and paralysed on the right side. After some days she could move her arm and leg quite well, but became deluded, noisy, and dirty in her habits. Six months later (June 1, 1934) she was admitted to the Fountain Hospital where her physical and mental state has remained unchanged. She is incontinent, and suffers from severe spastic paraplegia, with adduction spasm and contracture of both feet, more marked on the right side. The right pupil is larger than the left and does not react to light. She is observant, and appears to understand what is said to her, but she cannot answer simple questions, and talks mainly in parrot fashion. She shows definite facility of emotional reaction, and her general mental state is suggestive of an aphasia. There can be little doubt of a gradual deterioration since the age of 3, and none at all of a rapid retrogression since her cerebral attack following scarlet fever. The possibility that this attack was due to a scarlatinal cerebral lesion cannot be ignored, but such occurrences are of extreme rarity, and it is far more probable that it was in the nature of a sudden activation of her syphilitic process, possibly lit up by her recent illness. Despite the almost identical serological findings, this case is in direct contrast with the previous ones throughout its course.

The remaining three cases show a similar tendency to spasticity and deterioration.

Case VI.—A boy, aged 6, whose mother is tabetic. He walked normally, and was considered to be normal until the age of 2, but he has never talked. He is now an idiot, who cannot stand unaided and who has obviously deteriorated for some years. He shows slight hydrocephaly (skull circumference 21½ in.), a low-bridged nose, narrow palate, and slight spasticity of the legs. There are no ocular abnormalities or fits.

Case VII.—A girl, aged 14 years and 2 months, whose father died of G.P.I. and whose twin sister is mentally defective. She could walk until she was 3 years old; since then she has deteriorated. She was admitted at the age of 10 years, suffering from spastic paraplegia, pupillary defects, and *petit mal* attacks. The fits have increased in frequency, and she has become progressively more helpless and crippled.

Case VIII.—The last case is that of a girl, aged 17, whose mother is a tabetic. She walked at the age of 2 years, but never talked clearly. She began to "go off her feet" when aged 5, and was admitted at the age of 13½, suffering from spastic paraplegia, pupillary defects, choroiditis and slight optic atrophy. She has had no fits. She deteriorated mentally and physically, and died from a terminal bronchopneumonia in July 1934. The brain showed thickening of the meninges and perivascular infiltration of meningeal and cerebral vessels. There was, however, little derangement of the cell lamination of the cortex or other pathological lesions characteristic of G.P.I. The cord showed pyramidal tract degeneration. No spirochætes were found.

Except as regards their cerebrospinal fluid, these cases show little relationship to congenital G.P.I. or tabes; the onset is too early, the clinical picture is totally different, and the progress of half the cases is diametrically opposed to what we usually find in these conditions. It is true that pupillary abnormalities occurred in six of the cases, choroiditis in four, and slight optic atrophy in three, these signs being fairly

equally divided between the two subgroups. Fits, on the other hand, were present in only one case and in none was there any ataxia, tremors of lips or tongue, or speech defect of paretic type. The pathological findings in the one available case are consistent with meningo-vascular syphilis, but by no means typical of congenital G.P.I. It is true that the syndrome presented by congenital G.P.I. is far from definite, that many cases are mentally defective from birth and that the duration tends to be longer than in the untreated acquired form. On the other hand, the disease does not show steady amelioration over a period of years, nor, usually, the presence of gross cerebral motor lesions. Is it, then, justifiable to describe an infantile form of congenital G.P.I., whose main differences from the juvenile type are (1) earlier onset, (2) presence of cerebral diplegia and (3) longer course? For reasons to be stated later I do not think it is.

With regard to progress: four of these patients have shown steady improvement, while four have progressively deteriorated. In the latter four there would appear to be an activation of the spirochætal infection in the brain between the ages of 2 and 7, just as interstitial keratitis usually manifests itself during the next decade and juvenile G.P.I. during adolescence. In infancy there appear to be no criteria by which the ultimate progress of these cases can be foreshadowed. Of the deteriorating subjects all four were backward from birth, but all could walk by the age of 2. At different times during the next four years signs of pyramidal involvement became evident, together with definite mental and physical impairment. Although the onset of such signs may be taken to be of bad omen, their prognostic value cannot be considered absolute, as one of the improving cases shows a mild residual hemiplegia and paraplegia of spastic type. It is of some interest to note that the mothers of two of the deteriorating subjects are tabetics and that the father of another died of G.P.I. This coincidence of hereditary neurosyphilis has, of course, been observed before, but I do not think it will convince many of the existence of a neurotropic strain of *Treponema pallidum*.

Turning to the group of nine cases showing normal cerebrospinal fluids, I find it extremely difficult to decide whether all of them can justifiably be labelled neurosyphilis. Three of them suffer from spastic diplegia, and of these, two have progressively deteriorated, while one remains stationary; two have frequent and severe fits, and two show poor pupillary reaction to light. Although the possibility of a non-specific cerebral diplegia occurring in a congenital syphilitic must not be overlooked, I think one may fairly conclude that the neurological lesions and mental deficiency in these cases are due to the luetic process. Their history, clinical picture and progress are altogether similar to those of the deteriorating cases in the former group, and they might well be all classed together were it not for the differences shown by their cerebrospinal fluids. It does not appear reasonable to describe the former cases under the heading of an infantile form of G.P.I., and to exclude the latter merely because of serological differences. Nor is it feasible to include under such a heading the non-spastic improving cases, whose whole picture and progress are so dissimilar, on the bare grounds of their paretic type of cerebrospinal fluid. The cases are far too few, and the whole group beset with too many exceptions, to admit of any definite classification being formulated at present. At this stage we must inevitably find ourselves doubting the value of the cerebrospinal fluid picture in congenital syphilis as a guide, whether to diagnosis, prognosis or treatment. It is the practice at the Fountain Hospital, and at most mental hospitals, to lumbar-puncture all patients showing positive blood-Wassermann reactions, and I am eager to hear whether this procedure is carried out on congenital cases at any venereal clinics, where the figures would be very much larger and more impressive. Personally I should expect to find a paretic type of cerebrospinal fluid picture in a large number of congenital cases of all ages and conditions, but I am doubtful as to how much prognostic significance should be attached to such findings.

The remaining six cases show no ocular or central nervous system lesions commonly associated with syphilis. Three are young girls of imbecile grade who are definitely improving. They show syphilitic stigmata—e.g. bossing of the forehead and low-bridged nose—but also other stigmata such as epicanthus, squint, etc., and there are no signs specially indicative of cerebral syphilis. The seventh case is that of a feeble-minded girl aged 19½, who has improved steadily since her admission at the age of 4. She exhibits no stigmata or abnormal physical signs.

Are we to consider any of the last four patients, who are undoubtedly congenital syphilitics, but whose only neurological abnormality is mental deficiency, as examples of neurosyphilis? If we are to do so, the incidence of ordinary congenital syphilis at the Fountain Hospital falls far below that of the normal population, which is, manifestly, highly unlikely.

Of the last two cases, one is a very striking case of nævoid amentia, a condition not usually associated with syphilis, and the other is a typical cretin. Although endocrine disturbance is not uncommonly associated with congenital syphilis, I do not think that the thyroid deficiency in this patient or the nævoid amentia in the other case can be considered of more than coincidental origin.

The problem of deciding these points would be much easier if there were a definite mental syndrome attached to congenital neurosyphilis. I have tried to find some mental symptoms common to these cases, but have discovered little of definite value. The symptoms differ very much, according to the absence or presence of diplegia, the temperament of the diplegics corresponding closely with that of non-specific diplegia. It has struck me, however, that the syphilitic patients, whether diplegic or not, tend to show a greater emotional facility than other aments; they laugh immoderately for the most trivial cause, and are reduced to tears by the slightest discomfort or pain. They are of a timid and nervy disposition, and make a great deal of fuss at the slightest indication of any unpleasant attention. Change of mood is both frequent and rapid.

This facility is, of course, a prominent feature in general paresis, but it is present in some degree in most forms of cerebral disease. Unfortunately, there is no mental syndrome anything like so definite as can be observed in other congenital cerebral states, such as hydrocephaly, microcephaly, or nodular sclerosis.

Finally, with regard to treatment: During the last year all these patients have had arsenical and mercurial treatment, whilst the older ones have also had potassium iodide between courses. So far only one has shown any improvement which might reasonably be due to treatment, and in none has there been any serological change. The pyrexial treatment of cases showing a positive cerebrospinal fluid must now be considered. It is obviously contra-indicated in idiots or low-grade imbeciles showing long-standing spasticity. The improving cases, however, present a problem of some difficulty. Are we to give malaria to the girl of 19 who has grown from a sickly baby to a healthy working patient, in case one day her infection may flare up? If that calamity be considered a real danger, what should our attitude be regarding the woman of 25 whose cerebrospinal fluid also shows the changes characteristic of G.P.I.? At what age should we hold our hand in dealing with definitely congenital cases? With the example of these two cases before us, is it justifiable to tell the parents of the two boys of 10 and 11, who are now doing very well, that without some form of pyrexial therapy their prognosis is very grave? Until there is definite evidence that such cases do flare up or develop typical general paralysis or tabes, I do not think the induction of malaria should be recommended, but at the same time one cannot help viewing with some anxiety the progress of these subjects.

Mr. Lindsay Rea said that it was difficult for an ophthalmologist to contribute much on the subject of syphilis in neurology, but, from the negative point of view,

he might make some kind of addition to the discussion by means of some lantern illustrations. (Slides shown.)

He had never seen a central scotoma in a syphilitic case. Rayner Batten and himself had searched through hundreds of cases of macular disease, but out of all those examined, both toxic and familial, there was not one of a syphilitic nature.

A healthy man for whom he had prescribed glasses three months previously had come back to him saying that something was wrong with his vision. He had a crescent-shaped haemorrhage delineating the outward border of his macula. A month later the retinal arteries were still there, passing over the surface of the macula, but the hæmorrhage was beginning to change its appearance; pigment was beginning to appear and central vision was less than $\frac{6}{60}$, the finished picture being a scarred macular area, the same condition as had been in the other eye for over a year. It was a typical bilateral macular inflammation or degeneration. But never at any time is that appearance found in syphilis, either acquired or congenital. A skiagram of the teeth of this patient showed pockets of pus. There was severe diffuse osteitis, and another slide showed an old root which had been left in by the dentist. He (Mr. Rea) could scarcely be blamed for relating the two conditions.

The President had said that perhaps the spirochæte was aided and abetted by some other organism. It was many years since he (the speaker) had arrived at the conclusion that gonorrhœal iritis occurred only in persons who had some form of dental sepsis, and that the two were definitely related, and a recent writer also had pointed this out. He (Mr. Rea) was sure that many of the diseased conditions were not due to one specific organism, but that there was present some aggravating or abetting toxin, giving rise to the various manifestations. Syphilis, however, was a very definite disease. It had many manifestations, but any one beginning his career at hospital should make up his mind at once never to be careless or indefinite about his treatment of it.

[Slides were shown illustrating the difference in results in cases of interstitial keratitis which had not received specific treatment and those which had. Those fully treated with arsenical injections, together with mercury and iodides, gave by far the best results.]

Congenital syphilis should be fully treated. Jonathan Hutchinson had said that deafness came on in these congenital cases just as the interstitial keratitis was clearing up. He (the speaker) had had 300 cases of interstitial keratitis which he had had the persistence to treat, but had never seen a case of deafness yet, though many had been watched for as long as ten years.

If he (Mr. Rea) had been a young man just appointed on the staff of a hospital for the first time and had been listening to this discussion, he would have gone away with the question in his mind, "Is it worth while treating these syphilitic cases, whether they be nerve cases, or ophthalmological cases, or whatever they may be?" He had worked hard at this subject, and nothing would ever convince him that one's duty was other than to see that every case of congenital syphilis should have the benefit of full treatment, and treatment continued for at least two years. It required a good deal of both time and patience, but it must be carried out. Some of his own patients, after this treatment, were mothers of healthy children. One mother had been growing blind owing to gradually disseminating choroiditis, but treatment with mercury and iodides prevented the calamity.

Mr. Sydney Scott: It is extremely difficult to diagnose from clinical examination and hearing-tests whether an auditory defect in a given patient is due to a syphilitic lesion or not. Of course we occasionally see obvious syphilitic lesions in the external ear, but these do not come into the discussion.

I know no special otological sign in response to hearing tests which is pathognomonic of syphilis of the cochlear or vestibular nerve, yet there is, of course, no

question that we do meet with the disease. I have seen a child who had the stigmata of congenital syphilis—Hutchinsonian teeth and interstitial keratitis—who was deaf—absolutely deaf—in one ear only. The labyrinthine tests on the deaf side were all perfectly normal, showing that the vestibular division of the 8th nerve was unaffected. But on the other side, the right, in which there was perfect hearing, the vestibular tests produced no response whatever. The external and middle ears were quite normal on each side. The conclusion was that the left cochlear nerve and the right vestibular division of the right 8th cranial nerve were defunct, but we do not know the precise nature of the lesion.

The difficulties facing the otologist are great, because there are so few pathological otologists, and it is provokingly difficult to obtain material post mortem even in cases specially tested during life. Probably because the otologist is not "in at the death," and all corpses are deaf, no special attention is paid to the cochlea!

Moreover the examination of the cochlea is no easy matter. Even if we succeed in decalcifying the bone and mounting sections, it is impossible from just a few sections to examine the whole of the cochlea. Several hundred sections are necessary. They must also be as thin as possible, and orientation of the organ of Corti is often fortuitous.

[Mr. Scott showed on the screen a slide illustrating a section of the organ of Corti which he had obtained from a normal human cochlea an hour and a quarter after death. The section was cut in the axis of the hair cells, and one complete cell was seen, with nucleus and long cilia emerging.]

We cannot, however, always obtain a section showing the normal structure so clearly. Many sections contain artefacts, which have often been mistaken, we think, for lesions.

Dr. Gray has kindly lent me some slides from which I select one showing definite pathological changes in a deaf-mute, but there is nothing definitely characteristic of syphilis, though there may have been disease of the vessels in stria vascularis—which probably secretes the endolymph [slide shown]. No spirochætes have ever been found in the cochlea, I believe, and Dr. Gray will bear me out, but the difficulty of recognition, even if they were present, must be prodigious.

Turning back to the clinical tests of hearing, I need hardly say that the use of a single tuning-fork to test air and bone conduction is of little or no value in investigating the causes of nerve deafness. It is necessary to test the whole tone-range and to ascertain the lowest audible tone in terms of cycles or double vibrations, and also the highest audible tone. Whatever method is preferred, the most reliable is considered to be a series of tuning forks and the monochord.

[Mr. Scott here exhibited a slide showing the tone-ranges of a group of patients examined at the National Hospital, Queen Square, and drew attention to the losses of the upper tones. The cases included various forms of so-called nerve deafness indistinguishable from those associated with "senility," often premature.

He then showed a slide illustrating the results of tests in another group, characterized by losses of the low tones and of the upper tone limits. These cases included examples of auditory nerve tumours and other diseases. Only in the very deaf patients, where the remnant of hearing was represented by a short island in the tone scale, had syphilis been suspected, and of this, for the reasons which he had given, there were no proofs apart from blood-tests.]

A very rare condition would be noticed in a few of the cases illustrated, namely, dual remnants in the tone-range, that is to say loss of the lowest, highest, and some intermediate tones, leaving gaps and islands in the hearing range. Whether these islands indicate zonal disease in the cochlear nerve or in the nerve-endings we do not yet know for certain.

I feel justified in pleading on behalf of those of us who are interested in the association of clinical findings and pathology of the auditory nerve-endings, that the

internal ear should be removed as opportunities offer at autopsies and preserved for decalcification and examination, in order to help in elucidating certain of these unsettled problems in neurosyphilis.

Dr. Hugh Garland said that one point had not, he thought, been sufficiently stressed in the discussion, namely, the significance of a negative blood-Wassermann reaction *plus* a negative cerebrospinal fluid in cases of neurosyphilis. He had recently seen twenty-seven cases of what he called neurosyphilis because these patients had had some symptom, usually pain, with pupillary changes, loss of ankle-jerks or knee-jerks, or both, and a loss of the vibration sense in the feet, while all other recognized neurological conditions could be excluded. In five of the male patients there was no history of syphilis; they were all Wassermann-negative and had normal cerebrospinal fluid. The majority of the female patients gave no history of syphilis and their blood and cerebrospinal fluid gave negative Wassermann reactions. Some men gave a history of untreated syphilis twenty-six years before the onset of neurological symptoms, this being the average time. Most of these cases, under treatment, were improving, subjectively at any rate. He therefore considered that antisyphilitic treatment was a very important thing.

He would like, however, to quote a case which was somewhat against what he had been saying. It was that of a man, aged 31, who presented himself at Leeds Infirmary with a chancre, a generalized rash, and a positive Wassermann reaction; the cerebrospinal fluid was not examined. He had a course of 4 grammes of novarsenobillon together with bismuth, and the Wassermann reaction became negative. He was then given a rest period, followed by a further course of anti-syphilitic treatment. Towards the end of the second course he had a unilateral headache for which he was given aspirin tablets. At the end of the second course the Wassermann reaction was again negative. Three weeks later he was still complaining of unilateral headache, but he had no signs. The cerebrospinal fluid was examined by the speaker and it was strongly positive; there was a paretic Lange curve, 350 cells per c.mm., and the blood-Wassermann reaction was positive. Thus, this was a case in which the patient developed neurological signs in spite of anti-syphilitic treatment.

Dr. Fergus Ferguson said he wondered whether in the cases of early tabes dorsalis with lightning pains, absence of ankle-jerks, and some ataxia, one was justified, either as an initial form of therapy, or—if there was no improvement after six months' treatment with iodides—bismuth and arsenic, in advocating malarial treatment.

He also wondered, with regard to the differential diagnosis between neurasthenia and dementia paralytica, whether one could exclude general paralysis of the insane if the blood-Wassermann reaction was negative, or whether there had been proved cases of dementia paralytica in which there had been a negative blood-Wassermann reaction before treatment. Private patients did not think that it should be necessary to examine the cerebrospinal fluid to differentiate between these two conditions.

Dr. Leonard Findlay said he was stimulated to rise by the query of Dr. Ferguson, but before referring to the specific question regarding the malarial treatment of congenital neurosyphilis, he could not help remarking, with all due respect to the President, that he was astonished that anyone should, in the year 1934, discuss at such length whether syphilis, as met with in the child, should be called either "congenital" or "hereditary." He (the speaker) would have thought that the mere admission that it was of bacterial origin dismissed it entirely from the

field of heredity. Of the treatment of syphilitic meningo-encephalitis in the child he had had some slight experience. He had treated cases of that condition with mercury, and, later on, with salvarsan, both intravenously and intrathecally, but he had never seen any improvement result. He had also had the opportunity of treating seven cases with malaria, and he had not observed any special dangers associated with the method. In five cases no benefit resulted, but in one the disease was arrested and in one apparently cured. In both these latter cases the history of the trouble was of comparatively short duration, unlike the vast majority of cases of neurosyphilis in the child in which it was difficult to say when the disease had started. Often such children were found to have been backward since birth, and therefore one could not expect much from any line of treatment.

Mr. Leslie Paton said that in his opening paper the President had referred to the rarity of central scotoma in cases of neurosyphilis. So far as he (Mr. Paton) had been able to discover, there was only one reported case of a central scotoma in such disease, that by Wilbrand and Saenger, and even that seemed doubtful. On one occasion he (the speaker) thought he had a case of central scotoma in a tabetic. There certainly was a beautiful central scotoma, but it was not due to tabes, it was caused by tobacco.

There was a possible anatomical explanation for the absence of central scotoma. After having tested a very large number of fields in cases of tabes, one found that these cases fell into two classes. In the first the visual acuity diminished quickly, with no marked limitation of the fields at all in the early stages; there was a kind of uniform degeneration in the sensibility of the retina or of the activity of the optic nerve. That was the type where the parenchyma of the nerve was primarily affected. The second type of case was that in which, often, the central acuity remained good for a long time, and in which there was a very definite limitation of the fields, sharply-cut sectors lost, quadrantic hemianopia, sometimes complete bilateral hemianopia, and, occasionally, altitudinal hemianopia. In this type the invasion of the nerve was from the pial sheath and primarily interstitial. A further inference from examination of the fields was that the interstitial type mostly began intracranially and affected the chiasma or intracranial portions of the nerve first. It was an anatomical fact that the central retinal artery and retinal vein only entered the nerve about half an inch behind the eye, and the upper part of the nerve had no central vessels. Consequently, the axial fibres in this part of the nerve were most protected and only affected when the whole parenchymatous tissue of the nerve was attacked. He believed that both types of invasion occurred: parenchymatous invasion of the optic nerve, and the interstitial. The fields brought that out very clearly.

In answer to one positive statement made by Mr. McDonagh, he (Mr. Paton) had definitely seen a case of reinfection with syphilis. The man was a well-known sporting journalist, who was for some time under his (the speaker's) care on account of extensive syphilitic retinitis, which evidently responded well to treatment, as vision was finally very good. There was no doubt about the syphilis, as the man knew where and when he got the infection. He was then 56. At 65 years of age he got a fresh primary chancre.

Section of Surgery

President—PHILIP TURNER, M.S.

[*November* 7, 1934]

Unusual Perforations

PRESIDENT'S ADDRESS

By PHILIP TURNER, M.S.

"UNUSUAL perforations" is a very wide subject and I will say at once that I propose to confine my remarks very largely to my own experiences. It is first necessary to define the limits of our subject. Acute abdominal cases may be divided into two groups: (*A*) A group which may be called "abdominal emergencies," and (*B*) a group which may be called "abdominal urgencies." An "emergency" may be defined as an event calling for immediate action; while an "urgency" may be defined as an event calling for immediate attention. Thus an abdominal emergency is a condition calling for immediate or early operation which is necessary as a life-saving measure. The condition of the patient does not permit of any X-ray or other examinations or investigations and may even make it difficult to get a reliable history: hence the operation is often exploratory, so that the operator sometimes does not find the suspected lesion but has to search for some unsuspected and possibly rare cause, often under conditions of great stress and difficulty.

In an abdominal urgency though an operation may eventually be called for, it is not immediately indicated, and though the symptoms may be both acute and grave, there will be an interval for observation, examination, and investigation, which will usually allow an accurate diagnosis to be made.

The type of perforation to be discussed is that which will give rise to a true emergency as has been defined. There are many emergencies, but perforation of an abdominal viscus is generally characterized by a very sudden and acute onset, severe symptoms, collapse, and typical abdominal signs.

"Unusual perforations" suggests that there are also usual ones. Perforations of gastric and duodenal ulcers, and of the appendix may be termed "usual." Nothing further will be said about the appendix, since even the most acute lesions with obstruction, gangrene and perforation are rarely, if ever, as acute as a typical perforation of a gastric ulcer, and their diagnosis and treatment is generally straightforward.

Though most perforations of gastric and duodenal ulcers are equally straightforward, unusual and puzzling examples of these usual perforations are not infrequent. The following is a case of this description:—

The patient, a woman aged about 45, was admitted as an abdominal emergency. For two days she had suffered from generalized abdominal pain; she had vomited several times and the bowels had not acted. The abdomen was enormously distended; there was everywhere a tympanitic note on percussion, with no signs of fluid. The case was thought to be one of intestinal obstruction with general intestinal distension. The operation was performed by Mr. N. L. Eckhoff and it was thought that it would in all probability be necessary to tie a Paul's tube into the lowest of the distended coils. However, when the peritoneum was incised, there was a tremendous escape of gas, which was free in the peritoneal cavity, and the abdomen collapsed. The intestines were not distended, there was general peritonitis, though with very little fluid, and nothing abnormal could be found in the stomach, duodenum or appendix. The peritoneal cavity was drained but the patient died three days later. At the autopsy there was an ulcer in the deepest part of the duodenum, which had perforated, but the opening had been covered by lymph.

In another group of cases injury may be the actual exciting cause of the perforation and the resulting emergency may be regarded as rupture of an abdominal viscus. I once saw such an accident and was called upon to see and treat the man

afterwards. He was pulling a heavily loaded hand-barrow down the slope from the south end of London Bridge. As he was in front of the barrow and thus unable to use his weight as a brake, he lost control and the barrow ran away with him. In a last vain effort to avoid disaster he turned round and tried to push, but was carried on and pinned against a post by the barrow handle—which struck him in the umbilical region. Shortly afterwards I was asked to see him in the hospital. He was in great pain and the upper abdomen was rigid and very tender. Having seen the accident, I felt sure that there was a ruptured viscus and decided to operate at once. To my surprise I found a typical perforated ulcer, surrounded by the usual indurated and œdematous area. There was no hæmorrhage or laceration and, beyond the perforation, no evidence of injury was found. The perforation was closed and the patient recovered. Afterwards, when questioned, he gave a characteristic history of long-standing attacks of severe indigestion and there can be no doubt that he had a chronic ulcer which was unsuspected and possibly on the point of perforating, the injury giving the final touch. Twenty years ago there was a discussion before this Section on rupture of the intestine, and I find that I described a similar case [1].

I do not think that it is unusual for perforation of one of these chronic ulcers to be due to some sort of injury; not a severe injury such as those described but something much slighter, such as muscular action or an attack of coughing. For instance I remember a patient whose ulcer perforated as he was bending down to put on his boots.

We have now to consider certain ulcerations of the intestine which may perforate and cause an emergency strongly resembling perforation of a gastric ulcer.

Typhoid fever is now a comparatively rare disease and I am informed that even in fever hospitals cases of perforation are very seldom seen. A perforation occurring in the course of a case that has been diagnosed and is under treatment does not concern us, as there will be little doubt about an emergency which develops in these circumstances. But there used to be cases, and I suppose they may still very rarely occur, known as ambulatory typhoid, in which the symptoms were mild, often so mild that the patient continued to get about and even to work, and in which the first indication that anything was wrong was the sudden onset of all the signs and symptoms of an acute emergency. I have notes of two such cases, both over twenty years ago, and both very typical of this condition. One is of such interest that I will quote it in some detail.

The patient, a man, aged 46, was admitted under Dr. Hurst in 1912, on account of acute abdominal pain. His condition was very grave; indeed he was so ill that it was with the greatest difficulty that any history could be obtained. For two weeks previously he had suffered from abdominal pain, chiefly in the upper abdomen. The bowels had acted regularly and there had been no vomiting. For a considerable part of this time he had been unable to work and had rested in bed, but he had not seen a doctor. Three days before admission the pain had become much more severe and he had vomited several times during the preceding night. On admission the pulse-rate was 120; the abdomen was distended and did not move on respiration: above the umbilicus it was rigid and tender; liver dullness was absent. There obviously was general peritonitis, and in view of the history of indigestion and the situation of the pain, tenderness, and rigidity, a diagnosis of perforated duodenal ulcer was made. When the peritoneal cavity was opened pus and gas escaped and there was general peritonitis with matting and recent adhesions of the coils of intestine. Examination of the stomach and duodenum showed no sign of ulcer or perforation and the appendix was normal. The condition of the patient was so grave that no further search was possible. He died a few hours later. At the post-mortem examination numerous typhoid ulcers were found, chiefly in the ileum, one of which had perforated. Many of the ulcers were healing and hence one had to assume that the perforation had occurred during a relapse.

No one had suspected that this case might have been one of typhoid fever.

An important and not uncommon cause of an emergency is perforation of a diverticulum. This may arise in diverticulosis of the colon, and is due to ulceration of a concretion through the thin wall of the diverticulum. The symptoms of such

a perforation are acute and severe; the preceding symptoms are likely to be vague, and hence the true diagnosis is likely to be missed, and the case regarded as one of appendicitis or perforated duodenal ulcer.

I was once asked to see a man aged 63 who was staying at a London hotel. When I saw him he was so ill that it was difficult to get any history. However we managed to obtain a vague account of abdominal discomfort and "indigestion" extending over two or three years.

On the previous evening he had had an attack of such severe pain that he had no dinner and went to bed, the pain continued all night and he vomited several times; his bowels had acted normally on the previous day. In the early morning he called for assistance and a doctor was sent for who asked me to see him. The pulse-rate was then 120; the man was obviously in great pain, and the abdomen was rigid and tender. It was obvious that he had acute general peritonitis, but the cause of this was uncertain. It was thought to be either a perforated duodenal ulcer or appendicitis, probably the former. He was removed to a nursing home for operation. The abdomen was opened by a right paramedian incision and immediately the peritoneum was incised gas and foul-smelling pus escaped. Examination of stomach and duodenum failed to show any ulcer and the appendix was no more inflamed than the adjacent intestinal coils. On making a further search for the cause the pelvis was found to be a pool of pus through which at one spot bubbles of gas could be seen rising. With some difficulty the pelvic colon was brought into view and several diverticula containing concretions were seen and felt. At one spot a perforation was seen through which gas was escaping. This was closed by two layers of sutures, and the rather large wound was closed except at the lower end where a large tube provided pelvic drainage. The patient died three days later.

Though no fæcal concretion was found, I do not think that there can be any doubt that the perforation in this case was caused by ulceration of a diverticulum.

A perforation may occur in a Meckel's diverticulum. Various causes have been recorded—a typhoid ulcer, tuberculous ulcer, and ulceration set up by a foreign body. But a point of great interest is that in this situation perforation of an ulcer of the type of an acute peptic ulcer is sometimes met with. Meckel's diverticulum usually contains mucous membrane of similar structure to that of normal small intestine. Occasionally, however, gastric mucous membrane, containing oxyntic cells, is present; in other cases pancreatic tissue has been found [2], as was first described in this country by H. S. Clogg. The name "heterotopia" has been given to this very interesting condition of misplacement of normal tissues.

An interesting point is that perforation of a Meckel's diverticulum may occur in children. Perforations of any sort are very rare in children, a perforated gastric ulcer being almost unknown. Here is a case of a perforated peptic ulcer of a diverticulum in a boy aged $3\frac{1}{2}$ years.

W. B., aged $3\frac{1}{2}$ years, was admitted on account of severe abdominal pain and vomiting. There was no history of previous abdominal trouble, but about a year previously he had had three attacks of pneumonia in rapid succession.

The present trouble had begun at 4.30 p.m. on the day before admission with a severe and sudden attack of pain in the umbilical region. The child was put to bed and a dose of liquid paraffin was given, but the pain increased, and two hours later he vomited. Since then the pain had been continuous with repeated vomiting.

On admission, temperature was 102, pulse-rate 148, and respirations 50. The child looked extremely ill and appeared to be in great pain. There was generalized abdominal pain, tenderness, and rigidity, but little or no distension. Rectal examination was negative. There was obviously general peritonitis, but the cause of this was uncertain. The very acute and sudden onset, quite as sudden as the perforation of a gastric ulcer in an adult, made it improbable that the appendix was the cause. Before the operation I thought that it might be a case of streptococcal peritonitis. Immediate operation was indicated; there were no physical signs in the chest and the urine was normal.

The abdomen was opened by a right paramedian incision, and when the peritoneum was incised pus escaped, but no free gas. The appendix, which was inflamed and œdematous, but not more so than the adjacent coils, was removed. It was opened, but was certainly not the cause of the peritonitis. The small intestine was investigated, and nine inches from

the ileocæcal valve a Meckel's diverticulum, surrounded by thick lymph, and with one coil of bowel loosely adherent, was found. The diverticulum was about one inch and a half in length. About half an inch from its intestinal attachment there was a small perforation, an eighth of an inch in diameter. It was not indurated, and from the outside no reaction in the margin of the opening could be made out. The diverticulum was not provided with a fibrous band, and was hanging freely in the peritoneal cavity. It certainly was not a case of "diverticulitis," and there was no strangulation or obstruction of the gut. Clamps were applied and the diverticulum was excised, the opening into the ileum being closed by two layers of sutures. When the interior of the diverticulum was inspected it was obvious that there were two kinds of mucous membrane: a proximal portion, occupying about half the total length, continuous with, and similar in appearance to, the mucous membrane of the bowel, and a distal portion, thicker and of firmer consistence. The perforation, which was not surrounded by any thickening or area of inflammatory reaction, was actually in the intestinal mucous membrane, but in contact with the thicker distal portion. It had the appearance and characters of a perforated peptic ulcer.

Cultures of the pus give a mixed growth of *B. coli*, *Strept. brevis*, and *B. welchii*.

The diverticulum was examined histologically by Professor Nicholson, who reported that the proximal mucous membrane had the structure of intestinal mucous membrane, while the thicker distal portion had the structure of gastric mucous membrane.

The boy stood the operation well, but in the course of the next few days his condition became very grave, owing to pneumonia, with cyanosis and threatened cardiac failure. On the fourth day after the operation the wound gaped, exposing small intestine in its depths. As the condition did not permit of resuturing, the edges were drawn together by strapping, but nine days later a fæcal fistula developed. Several attempts were made to close the fistula but without success, and as soon as the condition of the patient permitted it, the diseased coil was excised and an end-to-end anastomosis made. Though this was much less difficult than was anticipated, it was more than the boy's feeble condition would stand, and he died two days later.

In these cases of heterotopia gastric mucosa hæmorrhage from the bowel is not unusual, and the absence of this or other previous abdominal symptoms is noteworthy.

We are not, this evening, able to discuss the pathology of this very interesting subject, but I must take the opportunity of referring to a paper by Mr. Harold Edwards [3] in which the pathological aspects are considered and a bibliography is given. Mr. Edwards records a case of perforation in a remarkable diverticulum of the small intestine which he regards as an instance of reduplication. Briefly the boy, who was 16 years old when the emergency occurred, had suffered from infancy from severe attacks of abdominal pain, vomiting and melæna. He was admitted as an urgent case, after a very severe attack of pain, for X-ray and other investigations. While he was actually in hospital the perforation occurred, and he was operated on by Mr. Edmunds, who found the perforation in a diverticulum 28½ in. long, which he excised. A Meckel's diverticulum was present on the portion of ileum excised: hence some other explanation was required. The mucous membrane of the proximal part looked normal to the naked eye. In the distal (free) portion the mucosa was thickened and raised to form small polypoid masses. The ulcer was situated in the distal portion, 3 in. from where it left the mesentery to become free. Histologically the surface layer consisted of columnar mucus-secreting epithelium. Beneath this was a closely packed mass of glandular epithelium consisting of cells which exactly resembled the central glands of the gastric tubules. At the periphery of many of these glands, between the central cells and the basement membrane were oxyntic cells. Heterotopia may thus occur in Meckel's diverticulum and also in other congenital diverticula and, though rare, is of definite clinical importance. I believe that all cases recorded have been in males.

Perforation may also occur in other rare intestinal diverticula. I have had no personal experience of these, but I remember a case recorded by K. M. Monsarrat [4] of perforation of a duodenal diverticulum which gave rise to an emergency, resembling acute cholecystitis. Histologically the diverticulum showed the structure of normal small intestine with villi.

Perforation occasionally occurs with a neoplasm of the intestine or even of the stomach. Perforation of a stercoral ulcer is not uncommon, but this, or perforation of a malignant ulcer, is probably a terminal event, and as the nature of the original disease is usually known, the perforation can scarcely be described as unusual. Rarely the existence of the growth may be unknown and an emergency of puzzling nature may be the result.

The following case of perforation of a carcinoma of the jejunum is of great interest, not only on account of its rarity but because the history and symptoms were so typical that, had it been less rare, a correct diagnosis should have been made.

The patient, a woman, aged 65, was admitted on account of severe abdominal pain at 10 p.m. on April 12, 1929. During the past six months she had had a number of attacks of fairly severe abdominal pain, coming on suddenly at irregular intervals, colicky in character, with no definite relation to food, without any vomiting, and referred to the umbilical region. After she had rested for a few hours the pain passed away and completely disappeared until the next attack, which might not occur for some weeks. No doctor was seen. The last of these attacks had occurred three weeks before admission. The bowels had acted well, with the help of occasional aperients. There had been no diarrhœa, blood, mucus, or other abnormality about the motions. She had lost weight slightly during the past few months.

At 6.30 p.m. on the day of admission she had a sudden attack of very severe and acute abdominal pain on the left side, extending from the loin to the groin with its maximum in the left iliac fossa. She vomited five or six times and sent for her doctor who at once sent her to hospital with the provisional diagnosis of renal colic. On admission she was in great pain, and the left half of the abdomen was tender and rigid. There was no enlargement of liver or spleen, and no abnormal lump could be detected. The bowels acted well that morning. Pulse-rate 80; temperature 99·4° F.

An injection of morphia gave her a fairly comfortable night, and on the following morning an enema, given as a preparation for X-ray examination of the urinary tract, produced a good and normal result. The skiagram was negative, and the only abnormality in the urine was a faint trace of albumin. Vomiting re-commenced at 10 a.m. and by 11 o'clock the abdomen was slightly distended and everywhere tender, with board-like rigidity. No lump could be detected, but on percussion there was an impaired note in the left flank. Vaginal and rectal examinations showed no abnormality. The pulse-rate had gone up to 102. The skin was very dark, almost suggesting jaundice, but the patient said this was her usual colour; the sclerotics were quite clear. A yellowish-green patch, suggesting a bruise, was noticed in the left scapular region. There was no history of injury, and the patient said that this was a "birth-mark" and had always been present. The case was certainly an emergency. When the patient was under the anæsthetic the rigidity disappeared and it was then possible to feel in the left iliac fossa a hard mass the size of a large egg, elongated and slightly movable. This was thought to be a growth of the colon or, just possibly, diverticulitis.

When the peritoneum was opened pus escaped, and there was diffuse suppurative peritonitis. The lump proved to be a carcinoma of the jejunum about 8 in. beyond the duodenojejunal flexure, which had perforated. The growth was freely excised and an end-to-end anastomosis made. Drainage was provided and the wound closed. For a few days there were hopes of recovery; but chest trouble developed and the patient died ten days later. At the post-mortem examination a localized collection of pus was found in the pelvis.

In this case the six months' history of colicky pains with loss of flesh should have suggested a growth, and the absence of obstruction taken with normal motions and a normal condition of the rectum should have suggested that it was in the small bowel.

Perforation by a swallowed foreign body is uncommon. Provided that they reach the stomach, these bodies, though of the most varied size and shape, are usually passed naturally. I have known even an open safety pin to pass through the alimentary canal of a child without causing any damage. Even if they remain in the stomach they may be retained for a very long time without doing any harm. These objects are often swallowed by people who are either definitely mentally deranged or of weak mind; others are swallowed unwittingly with food. Hence, rather surprisingly, in cases of perforation, the presence of the foreign body is usually unsuspected. I once

operated on a woman who for some days had suffered from numerous colicky attacks with vomiting. She appeared to have some sort of chronic obstruction and it was thought possible that a gall-stone of large size was traversing the small intestine. At the operation a large triangular fish-bone was found impacted in the ileum. There was no peritonitis, but the slight manipulation of drawing the coil to the surface was sufficient to force one of the sharp angles through the wall of the gut. The bone was removed and the patient recovered.

The following case of perforation of the pelvic colon was operated on by Mr. R. C. Brock.

A man aged 42 was admitted January 28, 1932 on account of very severe abdominal pain which had come on at 10.30 p.m. on the previous day. It commenced in the hypogastric region but soon became general.

There was no previous history of abdominal trouble but, owing to constipation the previous day, the patient had taken a dose of salts that morning and the bowels had acted several times.

On admission the pulse-rate was 84 and the temperature 98. There was general abdominal pain, tenderness, and rigidity, this last being particularly marked in the umbilical region and on the left side. There was obviously general peritonitis, the appendix being regarded as the most probable cause.

When the abdomen was opened pus and gas escaped. The appendix was healthy. The pelvis contained pus and fæcal matter and on palpation of the colon a foreign body was felt projecting near its termination. The pelvic colon was drawn into the wound and was found to be intensely inflamed. The perforation was in the middle of a short pelvic loop, on the left side near the mesentery. The foreign body, apparently a large fish-bone, was removed; it was about 2½ in. long. Around it was a gangrenous area about ¼ in. in diameter. The perforation was sutured and covered with convenient appendices epiploicæ.

A rubber tube was passed per anum beyond the line of suture and the wound was closed with free pelvic drainage. The patient had a rigor four days later, but after that he did well and made a good recovery.

Perforation of the stomach by a foreign body is very uncommon. Indeed, foreign bodies may be retained for a long time without giving rise to any serious trouble. A case in which a needle had perforated the stomach in two places has been recorded by Mr. Jennings Marshall [5] who removed successfully a large mass of foreign bodies, including hairpins, needles and a nail, matted together by mucus, from a patient who had some mental trouble.

A perforation of the urinary organs may sometimes be the unsuspected cause of an abdominal emergency. Such may occur in the bladder as the result of injury or ulceration in a pouch.

I once saw a remarkable case of rupture, apparently spontaneous but probably really traumatic, of a hydronephrotic single kidney into the peritoneal cavity. This occurred in 1915 and I showed the patient seven years later before the Clinical Section of this Society [6].

Perforations of the gall-bladder are generally unsuspected rather than unusual and I do not propose to discuss them.

An extremely important and easily overlooked perforation is that in the posterior fornix, in attempts to procure abortion. As the result of this there will be diffuse pelvic peritonitis spreading up to the general cavity. Nearly always in such cases a misleading history will be given, and a diagnosis of appendicitis, or of suppuration spreading from the tubes, may be made.

REFERENCES

1 *Proc. Roy. Soc. Med.*, 1913-1914 (Sect. Surg., 105).
2 *Reps. Soc. for Study of Dis. in Children*, 1908, viii, 221.
3 *Brit. Journ Surg.*, 1929-30, xvii, 7.
4 Id., 1926-27, xiv, 179.
5 Id., 1923-24, xi, 590.
6 *Proc. Roy. Soc. Med.*, 1922-23, xvi (Clin. Sect., 24).

Section of Ophthalmology

President—RANSOM PICKARD, C.B., M.S.

[*November* 9, 1934]

Left Divergent Concomitant Squint—Painful Left Eye. Mass projecting into Vitreous from Optic Disc.—HUMPHREY NEAME, F.R.C.S.

F. R., male, aged 21.

History.—In 1926 patient lost the sight of the left eye almost completely; he thought things looked blurred on this side and so shut the right eye. He is certain that the eyes were tested at school and that both could see well. Long history of headaches and vomiting (usually together, rarely vomiting alone) which began after a blow on the top of the head by a falling chimney-pot.

1927: Operation by Mr. W. Trotter (decompression, left temple), which stopped the vomiting. Headaches began again three months later.

History of present illness.—Headaches very severe. Last few months still worse, with vomiting, usually after most severe headache. During two weeks only free from headache one day.

Present condition.—R.V. = + 0·5 ax. 90° = 6/5. L.V. Hand movements at 1 metre.

Right disc: good physiological cup. Slightly blurred upper edge and moderately heightened colour; no change in this from July 1933, to November 1, 1934.

Left disc: mass of "fibrous" tissue projecting forwards to anterior vitreous from disc, and some fine floating strands in and around. Peripheral pigment degenerative changes, spots of pigment and pigment on retinal vessels. Some slight choroidal pallor. The white mass projecting forwards = + 9 dioptres. Vessels around disc = + 1 dioptre.

Superficial Corneal Opacities—O. GAYER MORGAN, F.R.C.S.

Male, aged 34. No trouble with his eyes until 1923, when he developed irritation and redness in both while in the Suez Canal on his way to Hong Kong. The condition became worse and he was admitted to hospital, where the diagnosis varied from iritis to conjunctivitis. There was a gradual improvement, but the eyes have never been comfortable since that time. In 1927 the patient was under an ophthalmic surgeon and was treated for three years with different lotions, injections, etc.

Present condition.—Vision has been sufficiently good to allow him to act as inspector of police for all these years, but there is much photophobia and lacrimation at times and the eyes feel gritty and irritable.

The main points of interest in this case are, naturally, those of ætiology and treatment. As regards ætiology: (1) The condition began when the patient was on his way to the East; (2) there is a nine years' history; (3) the condition of eyes has varied from time to time, but they have never been completely free from discomfort since the condition started; (4) vision has been reasonably good all the time; (5) cultures have been negative; no focus of infection has been found; there is no history of either malaria or dysentery; (6) there is no suggestion of a familial element.

On examining the corneæ the most striking points are : (*a*) The fine branching, star-shaped opacities (fig. 1) ; (*b*) these opacities are in the epithelium (fig. 2), which is raised up, and there is, in places, a defect in the surface, as evidenced by the fact that the opacities stain with fluorescine ; (*c*) there is no loss of corneal sensation.

Comparing this with other cases of like nature : It is obviously not neuropathic. It is not familial ; also in these cases the nodules are beneath the epithelium, either

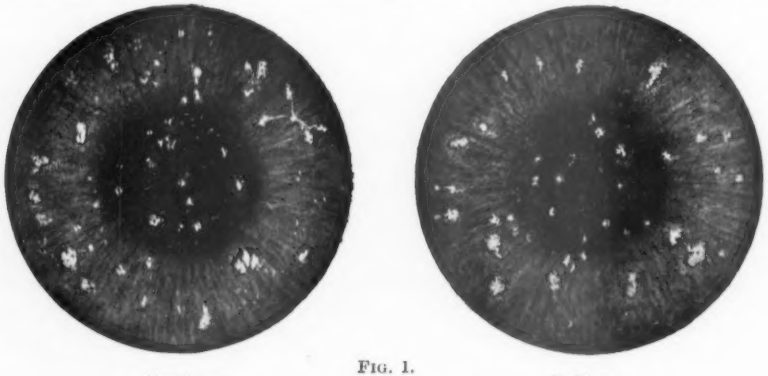


FIG. 1.

Right eye. Left eye.

Star-shaped epithelial opacities of the cornea.

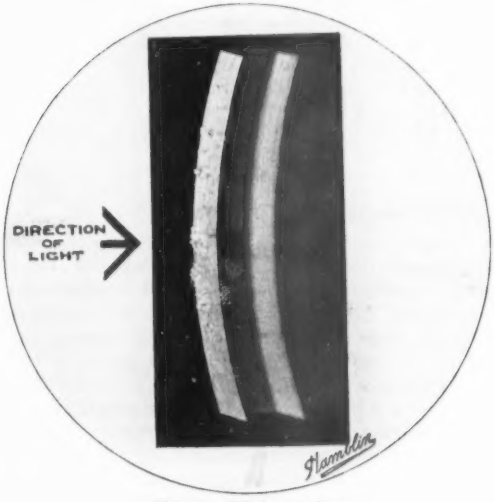


FIG. 2.—Slit-lamp view.

just deep to Bowman's membrane or in the anterior layers of the substantia propria. It does not belong to Salzman's group of nodular corneal dystrophy, as this is associated with chronic kerato-conjunctivitis, and there is a well-marked pannus. Fuch's superficial punctate keratitis is also associated with a catarrhal condition, the dots are beneath Bowman's membrane, and they disappear in from one to two years. Multiple erosions of a catarrhal herpetic type found in debilitated people stain with fluorescine, but do not last for years.

I can find no drawings of just such a lesion. I would call the condition multiple star-shaped epithelial opacities in the cornea.

With regard to treatment, the suggestions which have been put forward are (1) to scrape the surface of one eye, (2) to desquamate the epithelium by the use of chemical or light treatment.

Discussion.—Mr. J. D. MAGOR CARDELL said that in America about five years ago there had been published reports of cases of nodular keratitis in which a spatula could be placed beneath the edge of the nodules. The condition in those cases was, however, considered to be atypical.

Mr. F. W. LAW said that he had had a little experience of the condition. A suggestion as to treatment was that an overdose of ultra-violet light should be given to one cornea, so as to produce desquamation, and then see if the rejuvenated epithelium would show the same condition.

Neurofibromatosis of Orbit.—O. GAYER MORGAN, F.R.C.S.

This child, a boy aged 6½ years, was, according to the parents, normal at the age of 6 months, and a photograph confirms their statement. At about that age some facial asymmetry began to appear, and has progressed to the present condition. There is slight ptosis, but no loss of power in the upper lid, and no paresis of the seventh nerve on that side. Sensation is normal.

The eye is slightly proptosed, and is at a lower level. There is no limitation of movement in any direction. There is considerable bossing of the skull in both temporal regions, but the skiagram shows no abnormality except a slight displacement upwards of the left orbital roof. Cord-like structures can be felt in the upper lid, in the temporal region, and passing down in front of the ear to the angle of the lower jaw.

A familial element in these cases is quite common—e.g. in von Recklinghausen's disease—but there is no familial element in this case.

The coffee-coloured spots on the abdomen are typical, and the condition could be diagnosed on these alone. There are cases in which the body is covered with the pigmented spots, but in which there is no neurofibromatosis.

So far as I know, there is no recognized treatment for the condition, which is slowly progressive.

Vision in both eyes is normal, and the patient is quite bright intellectually.

Symmetrical Enlargements of the Lachrymal Glands. — T. H. WHITTINGTON, M.R.C.P.

Married woman, aged 51. Nothing of note in family history. First seen October 1931, complaining of aching pain located above and behind the eyes, intermittently for about four years, and recently redness and pain of the right eye. From about 1928 onwards, she had intermittent slight swelling of the upper lids and felt a lump behind the upper lid. She was found to have iritis in the right eye, and the condition was diagnosed as chronic adenitis of the lachrymal glands with iritis. The iritis settled down, but she continued to have neuralgic pains over and around the eyes with occasional slight œdema of the right upper lid. Thorough examination for local or constitutional disease proved negative. Lower or accessory portion of gland not involved either side. In March 1933 the solid hard masses felt at the outer half of the superior margin of the orbit seemed bigger, and in April 1933 I removed a piece of subconjunctival portion, and the pathologist reported chronic inflammation only. In September 1933 there was more swelling of the right side with some œdema of the lid. No effect on movements of the eyes, nor proptosis. In November 1933 I removed the right gland. It was very hard and cut like a scirrhous mammæ. It came out fairly easily by blunt dissection. A complete blood-count made two or three weeks later showed no abnormalities.

During the last few months the patient has had increasing ache in the region of the left gland, and, during the last few weeks, slight œdema of the lid.

Microscopical sections of the right gland show fibrous tissue mainly, with some scattered islets of masses of small round mononuclear cells. These islets of cells appear to be mononuclear lymphocytes, described by Dr. Knott as lymphomatous areas.

Vision in the right eye, with glasses, is $\frac{6}{12}$, $\frac{5}{9}$ part, and in the left eye $\frac{6}{5}$. Vision of the right eye is affected by a corneal opacity with anterior synechiæ, the result of a severe septic ulcer. This was before the gland was removed.

What is the ætiology and pathology of the enlargements in this case? Chronic inflammation or new growth?

Chronic dacryo-adenitis is, I think, very rare; the acute form is, of course, well known, associated particularly with mumps and less frequently with gonorrhœa.

Symmetrical enlargement of the lachrymals with enlargement of the salivary glands (Mikulicz's disease) is well known. There has never been any enlargement of the salivary or lymphatic glands. Tuberculous tumours of the lachrymal gland have been described, but in these cases tuberculosis is stated to be almost always present elsewhere.

Enlargements due to syphilis have also been described, but in both tuberculosis and syphilis chronic cases have been unilateral.

Uveo-parotitis is well known. This patient, probably for some long time after the beginning of the gland enlargement, had iritis, and it was partly this which brought her to hospital. Is there a "uveo-dacryo-adenitis" syndrome?

Tumours of the lachrymal gland have been described as endotheliomata, and mixed tumours somewhat like those occurring in the parotid, and also probably endotheliomata, or as Collins and Mayo prefer, tumours of mixed mesoblastic tissue; also lymphoid tumours of various kinds, and sarcomata. These appear relatively the commonest. The sections appear to show fibrous tissue mainly, with some lymphomatous areas. This is how Dr. Knott, pathologist at the Royal Waterloo Hospital, where the patient has been under my care, describes them. He does not think there is any sign of malignancy.

Can the case be one of lymphomatous growth of unusual chronicity, giving rise to irritation of the surrounding gland, and gradually causing its replacement by fibrous tissue?

No essential lachrymal gland secreting cells appear to be left, and there is nothing in the section to show that it is lachrymal gland.

The long history and clinical findings being against malignancy, and therefore complete removal being likely, I decided to operate. I think I was also led to this decision by the enlargement of the other gland, an additional reason for getting a section. The patient has now more aching pain on the other side and some œdema of the lid, and the hard mass behind the lid appears to be larger. The condition on the left side is much the same as it was a year ago when I removed the right gland.

Discussion.—The PRESIDENT said that outside the cases with full symptoms, one might meet with any single symptom, or any two symptoms together, without the usual grouping. The fact that the uvea was affected would lead him to include the case in the group of uveo-parotitis.

Mr. R. AFFLECK GREEVES said that judging from the sections projected on the screen, he thought the appearance was strongly suggestive of a chronic inflammatory condition.

Rupture of Descemet's Membrane from Contusion Injury in an Adult.—F. A. JULER, F.R.C.S.

D. M., male, aged 39, attended hospital in 1930 and again in November 1934, having symptoms of conjunctival irritation. The condition of the left eye was noticed in routine examination. He dates it from a war injury, in which he was thrown from a car by a bursting shell and was much bruised on the left side of his face.

Present condition.—L.V. $\frac{6}{6}$ partly (refraction + 0·5 cyl. ax. 45°). The left cornea shows a sinuous line on its posterior surface, extending from 10 o'clock to 5 o'clock, but not reaching the limbus. From this a "shelf" projects back into the anterior chamber, evidently formed by Descemet's membrane. This shelf is somewhat thickened and rounded at its free end, and there is a thinning of the posterior layer of the cornea corresponding to the previous bed of the shelf. There is some scattered pigment along the attached border, and there are in two places some rounded globules growing from the posterior corneal surface in the "shelf" area.

In birth-injury cases which I have seen, the astigmatism has always been high (8 or 10 dioptres), and the eye has been amblyopic. I have seen cases of buphthalmos in which a shelf has been formed, while in other cases the rupture does not take that form; the membrane splits and does not come away from the cornea.

Mr. A. C. HUDSON said that many years ago he had shown a similar case.[1] There were ruptures in Descemet's membrane, with a high degree of astigmatism, and there was evidence of good unaided vision not long before the accident.

Two Cases of Congenital Absence of External Rectus and Globe in Adduction.—R. COLSTON WILLIAMS, L.R.C.P., M.R.C.S. (introduced by Mr. AFFLECK GREEVES).

These cases are from Mr. R. Affleck Greeves' Clinic at the Royal London Ophthalmic Hospital.

One patient is a girl, aged 16½ years, and the other is a child aged 18 months. Both show the same anomaly of the left eye. There is defective abduction, and in addition, the palpebral fissure on the affected side widens when abduction is attempted and narrows on adduction, the latter being accompanied by retraction of the globe.

The two cases differ in that the baby's eye is straight in the primary position, while the girl has a superadded convergent strabismus of the affected eye, with amblyopia. The condition is not common, but is to be found described by several authors in the literature, the most complete account discovered being by Rudolf Aebli published in the *American Archives of Ophthalmology*, 1933, n.s. 10, p. 602. He mentions all the features found in these two cases and, in addition, notes that oblique upward and inward movement usually takes place on attempted adduction, while no movement whatever occurs in convergence.

Mr. AFFLECK GREEVES said he had seen a fair number of cases similar to these. All that he had seen had shown a deficiency in outward movement and all had this peculiar syndrome, namely, closing of the palpebral fissure on adduction and opening on abduction. He had regarded the closure on adduction as a retraction of the eye into the socket and the opening on abduction as an extrusion of the eye. In some of these cases, as in one of the two just shown, an ordinary concomitant convergent squint developed in addition; here the squinting eye was the one in which the muscle was deficient, though that was not always so. In the cases in which a concomitant squint did not develop, the eye remained straight in the primary position. Of these two cases, the girl has a convergent squint and an amblyopic eye, the eye with the deficient movement being the amblyopic one, but the child's eyes are straight in the primary position. He had followed the course of one case from infancy; she was now 19. The eye was straight in the primary position to begin with and then developed a convergent squint. She also had considerable hypermetropia, and glasses straightened the convergent squint, but she still of course shows inability to move the eye outwards and has now learned to turn her head round when she wants to look towards that side, so that the motor deficiency is less evident.

The condition must be due to a developmental error but it is difficult to say how it happens. In the human subject the development of the ocular muscles takes place from a sheet of mesoblast which envelops the eyeball, all the recti muscles becoming differentiated at the same date. He had never operated on any of these cases because all those who developed a concomitant squint had responded to treatment with glasses. The girl just shown had,

[1] *Trans. Ophth. Soc. U.K.*, 1920, xl, 214.

however, passed the age at which one would expect glasses to straighten the squint and he proposed to tenotomize the internal rectus with a view to straightening the eyes in the primary position. He hoped at the same time to investigate the state of affairs on the outer side of the eye with a view to determining whether there was an external rectus present or not, or any substitute for it.

(The President asked that the case be brought after the operation.)

Pre-operative Preparation of the Conjunctival Sac by Ultra-Violet Rays.

By Frank W. Law, F.R.C.S.

I have been asked to give a few details of the treatment, by ultra-violet irradiation, of those patients who, awaiting an intra-ocular operation, harbour on their conjunctivæ organisms which experience has taught us are potentially pathogenic if introduced within the eye. It will be noticed that the expression used in the title of my paper is not *sterilization*, but *preparation* of the conjunctival sac. The reason for this is twofold: First, it is not necessary to destroy or remove all the conjunctival flora before operating with impunity; secondly, it is unusual for irradiation of the conjunctiva to result in a sterile culture, the pathogenic organisms being apparently more susceptible to the influence of this agency than the non-pathogenic.

One of the most important points which I desire to make at the outset is that in the great majority of the cases referred to me for the purpose under consideration, all the ordinary methods of pre-operative preparation have failed. In all but a few cases the undesirable organisms have resisted the action of antiseptic lotions, and in many cases silver nitrate has been used, in an effort to render the conjunctiva surgically clean. It is, I submit, important to bear this fact in mind when assessing the results, as it favourably affects an estimation of the value of this form of therapy.

Technique.—The apparatus used is the modified general phototherapy mercury-vapour lamp which I have already described elsewhere. It consists essentially of a lamp mounted on a movable stand in front of a hemispherical housing, over the aperture of which is fixed a flat aluminium disc, pierced in the centre by a director tube. The patient's head is secured in the usual form of chin and head rest, and the lids are everted and held so as completely to cover the cornea. Though this may sound at once crude in idea and uncomfortable in execution, it is nevertheless quite comfortable for the patient, and satisfactory in practice.

The routine adopted is to give six doses of five minutes each at two-day intervals, irradiation being carried out at a distance of about twelve inches; the number of doses is increased if necessary. It should be mentioned here that it is quite as satisfactory—indeed, perhaps more so—to use the concentrated beam of the local phototherapy apparatus, with a suitably adjusted aperture.

For the present purpose I took at random the case-records of twenty patients who had been referred to me for pre-operative preparation. These cases fall for descriptive purposes into seven divisions.

(I) Eight cases are included in this division. In all of them the pathogenic organisms were exterminated, and the result of operation was normal as far as this investigation is concerned—by which I mean that convalescence was uneventful and their stay in hospital was not prolonged beyond the routine time. One of the cases (No. 3) had shown pneumococci and *S. aureus* in cultures over a period of six months, for which the normal measures had been adopted. Thirteen exposures succeeded in rendering the eye safe for operation.

(II) This group of three cases is similar, in that treatment was successful, but differs in the fact that the patients had, up to the time of writing, not been operated upon.

(III) Two cases are included here. In each case the conjunctival sac was rendered sterile, and in each case circumstances demanded a delay before operation

was undertaken. After this period of delay, a second culture showed that the sac was contaminated by the same organisms as before. In one case a further course again rendered the culture sterile; the other case was not so submitted. These results suggest the possibility that irradiation treatment does not actually destroy the organisms, but only attenuates them or inhibits their growth in some way, after which operation may be safely undertaken; an alternative explanation is that the conjunctivæ were reinfected.

(IV) This division includes two cases only; both patients ceased attendance after two exposures. The cultures in each case showed pneumococci, but both patients were operated on in spite of this; one had a normal convalescence, but the other spent a month in the ward, iridocyclitis with keratitis punctata following the operation; the end-result was, however, good.

(V) Again two cases are included in this group, and in each, in addition to the physico-therapeutic preparation, irrigation with oxycyanide lotion was carried out, and in one case silver also was used. The eyes proved to be intolerant of such a concentrated attack, and became red and irritable; the patients were discharged without operation.

(VI) This group is represented by only one case. Though the final culture showed no pathogenic organisms, operation was followed by an irritable eye, with cells in the anterior chamber. The case responded to treatment which included the administration of novarsenobillon; capsulotomy was successfully undertaken later, and $\frac{6}{18}$ vision resulted.

(VII) The last group includes two cases in which no apparent effect could be produced on the conjunctival flora. Both were, however, operated upon, with a "normal" result.

The results above described are summarized in the following tables. The reason for the treatment requested is given in column 2; the result of cultures before and after treatment in columns 3 and 5, and the number of doses in column 4. Figures in brackets indicate successive cultures. The following abbreviations are used:—

Pn.B., Pneumobacilli; Pn.C., Pneumococci; X, *Bacillus xerosis*; Aur., *Staphylococcus aureus*; Alb., *Staphylococcus albus*; Cat., *Micrococcus catarrhalis*.

TABLE I.

Case	Reason	Culture	Doses	Culture	Result
1	Extraction	Pn.B., X.	7	Alb.	Normal
2	Extraction	Pn.B.	6	Alb.	Normal
3	Extraction	Pn.C., Aur. Alb., Cat., X. (for 6 months)	7-6	Alb.	Normal
4	Needling	Aur.	6	X.	Normal
5	Needling	Alb., Cat. Pn.C.	7	X.	Normal
6	Extraction	Pn.C.	6	X.	Normal
7	Needling	Pn.C.	6	(1) Alb., (2) Alb., (3) Sterile	Normal
8	Needling	Aur.	9	Alb.	Normal

TABLE II.

Case	Reason	Culture	Doses	Culture	Result
9	Extraction	Aur.	6	(1) Sterile (2) Sterile	Awaiting operation
10	Extraction	Aur.	12	X.	,,
11	Needling	Aur.	6	Alb.	,,

TABLE III.

Case	Reason	Culture	Doses	Culture	Result
12	Extraction	Pn.C.	6	Sterile	
		One month's interval			
		Pn.C.	8	Sterile	No record
13	Extraction	Aur.	6	Sterile	Awaiting operation
			Interval		
		Aur. (2 colonies)			

TABLE IV

Case	Reason	Culture	Doses	Culture	Result
14	Extraction	Pn.C.	2 only	Pn.C.	Normal
15	Extraction (Chronic conjunctivitis)	Pn.C.	2 only		In hospital one month, with K.P. End-result good.

TABLE V.

Case	Reason	Culture		
16	Extraction	Pn.C.	Oxycyanide and silver used. Operation postponed	Intolerant
17	Extraction	Aur.	As above	

TABLE VI.

Case	Reason	Culture	Doses	Culture	Result
18	Extraction	Aur., Pn.C.	9	Alb., X.	Good—but in hospital 17 days

TABLE VII.

Case	Reason	Culture	Doses	Culture	Result
19	Extraction	Aur., X.	6	(1) Aur., Alb., X. (2) Aur., Pn.B., X.	Normal
20	Extraction	Pn.B.	5-6	Pn.B.	Normal

The conclusions which I draw from these results are as follows:—

(1) Ultra-violet irradiation is successful in nearly every case in rendering the eye bacteriologically safe for operation, even when the other methods fail.

(2) The pathogenic organisms appear to be more susceptible to the rays than the non-pathogenic, as evidenced by the infrequency of a resultant sterile culture.

(3) Some results suggest that the pathogenic organisms are only temporarily attenuated or inhibited, but that when this has been achieved operation may be safely undertaken.

(4) It is inadvisable to combine with this treatment the use of antiseptic lotions, and silver, as the eye appears to be intolerant of such vigorous measures.

I must express my indebtedness to the honorary surgeons at the hospital who refer their patients to me for this treatment, and to Dr. S. H. Browning who carried out the bacteriological examinations.

References.—Law, "Ultra-violet therapy in eye disease," London, 1934; id., "The present position of ultra-violet therapy in eye disease," *Trans. Ophth. Soc. U.K.*, 1933.

Discussion.—Mr. A. C. Hudson said that two years ago in his Presidential Address he had referred to an article by Montague Harston[1] on the effect of tungsten light on the flora of the conjunctival sac and his own results from this form of treatment, carried out by Dr. P.

[1] *Brit. Journ. Ophth.*, 1931, xv, 717.

Bauwens at St. Thomas's Hospital, as a preliminary to operation. His impression as to the value of the treatment, expressed at that time, had since been confirmed, particularly in the case of *Staphylococcus aureus* infections, such as had formerly proved extremely intractable to other forms of treatment. He had, however, had the same experience as Mr. Law with regard to the reappearance of this organism three or four days after an apparently successful treatment.

In his cases the irradiation had been applied through the closed lids, and not directly to the conjunctiva, and he thought that it was a case, not of killing the bacteria by the light rays, but rather of producing some stimulus of the tissues which enabled them to cope with the infection, and so get rid of dangerous organisms.

Mr. G. HANDELSMAN said he had treated in this way a number of cases of blepharo-conjunctivitis which did not respond to ordinary antiseptics. One patient had had the ultra-violet light beam applied for four minutes, to both upper and lower lids. Each lid was divided into three sections, and each section was irradiated for four minutes. After two such applications all the cultures were negative.

Another patient had come to hospital with cataracts, and pneumococci had been present in both eyes. He (the speaker) gave eight applications of the ultra-violet light, after which the cultures in that case also were negative.

Mr. D. V. GIRI: Mr. Law's method of preparing the eye for operation is to evert the upper lid and expose the conjunctiva in such a way as to hide the cornea completely and then irradiate with ultra-violet rays; thus the folds of conjunctiva in the upper fornix escape the direct action of the rays altogether. Mr. Hudson says that sterilization of the conjunctival sac has been achieved by ultra-violet irradiation of the closed eye. As ultra-violet rays do not penetrate deeper than the epidermis, it seems to me that in either case the irradiation brings about destruction or attenuation of the bacterial flora of the conjunctival sac by producing leucocytosis and phagocytosis rather than by direct action. As an animal host is essential for the viability of the pathogenic bacteria concerned, their reappearance within a few days of "sterilization" is probably accounted for by some of them in the folds of the upper fornix escaping even the indirect action of ultra-violet radiation.

Mr. LAW (in reply) said that Mr. Hudson had misunderstood his (the speaker's) remarks about treatment; the lids were everted and were so held as to cover the cornea when the light was applied. He had had no experience of irradiation through the closed lids; as far as his knowledge went, penetration of ultra-violet rays did not occur through the skin, and he did not know how irradiation through the closed lids would affect the conjunctival flora.

[Mr. HUDSON: My point was that it is not necessary to evert the lids at all.]

Mr. Giri had mentioned the action being indirect. He (Mr. Law) irradiated the lower fornix; the upper fornix did not, in his view, need irradiation, as it was seldom that any organisms were there.

He did not know how the rays acted. It might be that they caused a leucocytosis, and that this killed off the bacteria. It might be the direct action of the light which caused the cleansing of the conjunctival sac.

A Successful Corneal Graft with Demonstration of the Case.—J. W. TUDOR THOMAS, F.R.C.S.

A record of experimental work on corneal grafting in rabbits was published in 1930 (*Trans. Ophth. Soc.*, 1930, l, 127, and *Lancet*, 1931 (i), 335) and the most successful technique has since been applied to the treatment of cases of dense corneal opacities. In 1933 a paper was read before this Section,[1] on a successful case in man; the result in that case still holds good after four years.

At the Oxford Ophthalmological Congress in July this year, a case was demonstrated of a successful corneal graft on each eye, the first to be shown in this country, and, as far as I know, the first in the world. This patient—a lady—was recently demonstrated at a Congress in Boston, U.S.A.

The case now shown represents my fifteenth corneal graft in man, and as the patient will be returning to his home in New Zealand, this opportunity is taken of demonstrating the case while he is still in England.

The patient (Mr. B.), aged 57, suffered, at the age of 17, from severe corneal

[1] *Proceedings*, 1933, xxvi, 595 (Sect. Ophth., 33).

ulcers which left him with central corneal opacities and very defective sight. Some seventeen years later peritomy was performed on each eye. About twenty years ago the eyes became ulcerated again and from that time his vision was about the same as when he was examined in April of this year. The right eye was the better seeing eye. The Wassermann reaction was negative.

Condition before operation.—Figure 1 shows the condition of the corneæ. The right eye showed a dense central corneal opacity with considerable haze of the surrounding cornea, and some degenerative epithelial changes in the opaque area. The pupil was small, anterior chamber normal, projection good, and vision finger-counting at 15 in.

The left eye had still less vision, which was only hand-movements. There was a white extensive central leucoma of the cornea, with some superficial blood-vessels passing on to it and a few small interstitial blood-vessels. The periphery of the cornea showed hazy opacity. Projection was good, tension normal, and the anterior chamber was normal.

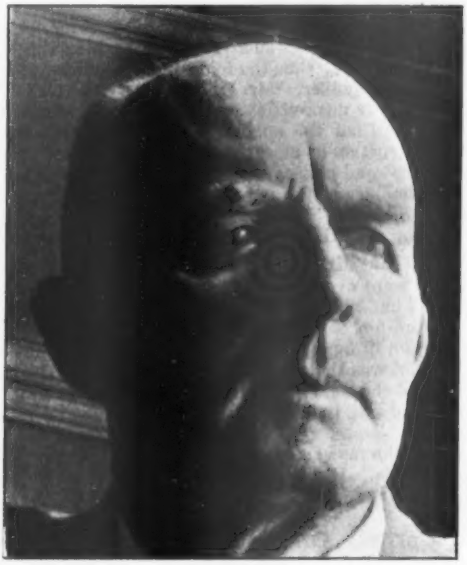


FIG. 1.

The patient had to be led about, unless he was in a place where he was quite familiar with his surroundings. He used to write on a slate having two rubber bands stretched across it to guide his pencil. He was brought to me as a private patient by his sister, who is a doctor in practice in London. It was decided to graft the left eye with new corneal tissue.

Operation.—The operation was performed at the Central London Ophthalmic Hospital on June 29, 1934 (about four and a half months ago). After a period of waiting, a donor eye became available from a patient, aged 60, of Mr. Mayou's, whose right eye had been excised on account of a choroidal sarcoma.

The technique of the operation was similar to that described in the experimental grafts, which gave the best results. A portion, about 4½ mm. in diameter, was removed from the cornea of the donor eye (after excision) by means of a trephine

and scissors, the margin of the graft being cut in a shelving manner and the graft placed in a watch glass of sterile olive oil.

Figs. 2 and 3 are microphotographs showing the appearance of the donor cornea after removal of the graft.

A larger trephine ($4\frac{2}{3}$ mm.) was used to outline the piece to be removed from the middle of the opaque cornea, and two cross sutures were inserted. The removal of the opaque piece of cornea was then effected by trephine and scissors, the margins being cut shelving. Citrated saline and adrenalin were used to ensure freedom from

FIG. 2.

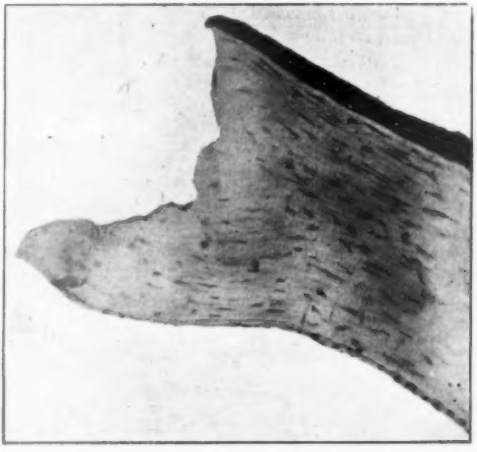


FIG. 3.

adhesion of the iris, as was described in the *British Journal of Ophthalmology*, 1934, xviii, 129-142.

The leucoma consisted of comparatively soft tissue and was not very vascular.

After removal of the piece of cornea the pupil was seen to be small and in the centre of the corneal opening. The iris was grey in colour and the lens clear.

The graft was laid on, the sutures were tightened and tied, 1% hyoscine in olive oil was applied, and a Cartella shield placed on the eye, with gauze on the anterior surface.

The man was a good patient and the operation was performed under cocaine anæsthesia with premedication by nembutal.

Figs. 4 and 5 are microphotographs of a section of the opaque piece of cornea removed. There are calcareous subepithelial deposits, but very few demonstrable blood-vessels.

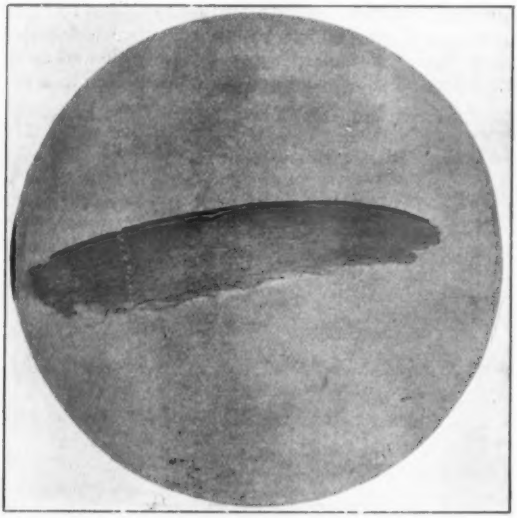


FIG. 4.

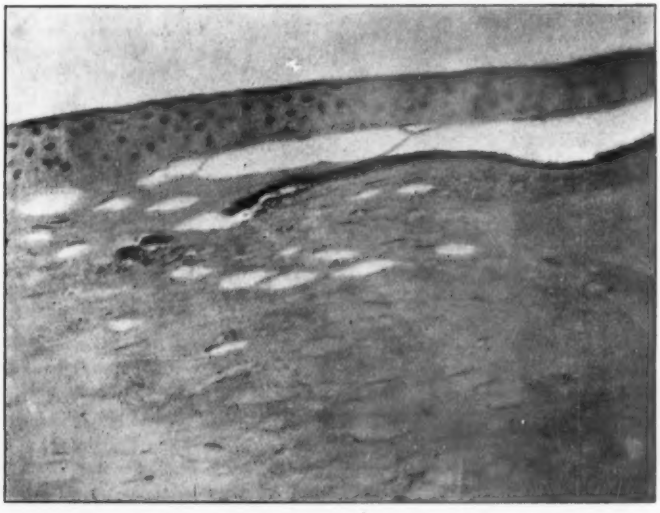


FIG. 5.—Showing subepithelial calcareous deposits in the piece of opaque cornea removed.

Progress after operation.—The eye progressed well, without pain. There was no redness of the lids, and only a slight amount of soft mucoid discharge.

The stitches were removed on the eighth day. The graft was well in place, clear and united, flat and round, and the eye was hardly at all red.

At fourteen days the graft was clear, with one minute white spot on the posterior surface near the lower margin, and a minute oily globule near the outer border. The shelving margin could be seen through the graft and in this shelving margin a few small blood-vessels took a circular course around the graft.

The graft was clear, with a smooth shining epithelial surface, and the pupil was easily seen through it.

From the eighth to the twentieth day the eyes were covered by pads and bandage, and after that dark glasses were worn, at first for short periods.

At three weeks the oily bubble had practically gone but the white dot persisted. The patient left hospital in a little over a month. On the thirty-third day his vision was one-sixtieth, on the thirty-fourth day two-sixtieths and on the thirty-fifth day three-sixtieths.

About this time he declares he could see Big Ben from half a mile away, and was getting much enjoyment by reading at a distance of two yards such notices in shop windows as " This shop is closed."

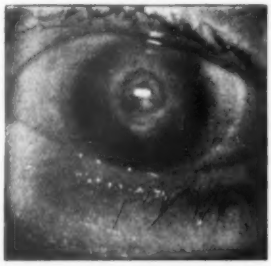


FIG. 6.—Four months after corneal grafting, left eye: the iris can be seen through the graft, and the pupil which is small, lies behind the light reflex.

He could read, at reading distance, the 6/5 letters on a Snellen Test Type Card, could negotiate street pavements and curbs, and could see a road island from the pavement. He could read the main headings of newspapers, and could pour tea and see things on the dinner table. His main hobby, however, was to read shop notices and the words on street posters. He was much relieved to be told by his friends that he had lost the vacant look of a blind man. He could tell the time by his watch.

At seven weeks his vision was between three-sixtieths and four-sixtieths. He had taken to playing cards. As a test he was given half a pack of cards to name each card and took forty-seven seconds to name the twenty-six cards, making three mistakes. The three mistakes were to name three cards " hearts " when they were " diamonds."

Present condition.—The condition at present (*see* fig. 6) four and a half months after the operation is that he can see four-sixtieths, or $\frac{6}{60}$, and has been for the last two months going about London by himself.[1] A more recent hobby has been, in addition to seeing London, to read the number plates of motor cars as they passed by; this he can frequently do.

The graft is clear, with a small opacity near the lower margin, which the slit lamp shows to be on the posterior surface, with a somewhat similar, but quite

[1] *Postscript.*—Tested a few days after this paper was read, his vision with +4·00 D. spherical was $\frac{6}{6}$.

separate, small white nodule on the iris below the pupil, suggesting that the two were in contact at an early stage after the operation, when the anterior chamber was very shallow. This small opacity—not 1 mm. long—is becoming less, rather than increasing. Slit-lamp examination shows the graft to be similar to normal cornea, with one or two horizontal grey lines in it.

The patient suffers from some increased blood-pressure and it has been noted that if the blood-pressure becomes much raised, the eye becomes somewhat red. The blood-pressure and redness of the eye subside quickly with a couple of days' rest.

It is, I think, fair to describe this case as one of a successful corneal graft.

I propose at a later date to make a full report on all my cases of corneal grafting, but at the moment the following statement summarizes the results.

Sixteen cases have been treated by transplantation of cornea, one of them only a week ago. Of the remaining 15, five were not really suitable cases and the results of these—not all failures—will be described in detail and are of great interest. In all the 10 suitable cases the graft became united. In the case of one of them the graft was successful, but the pupil was small and did not correspond with the centre of the graft ; this case is not yet completed. Seven others have been what may be termed "successful"—the standard of success being considerable improvement in vision, the graft retaining a considerable and lasting degree of transparency.

[The patient was then demonstrated to the meeting.]

Dr. MARY A. BLAIR, sister of the patient, said that, in response to the President's invitation, she would like to give members some idea of the improvement in her brother's sight, general condition, and outlook on life since the operation. From having little more than bare perception of light he was now able to play cards, even in artificial light. He suffered from very high blood-pressure, sometimes up to 220/130. It was noticeable that when his blood-pressure was raised from any cause, his newly acquired vision was not nearly so good. He could now find his way about London alone, and read posters as well as the headlines of newspapers. In a good light he could read type of one quarter inch or rather less at a distance of twelve inches from the eyes. He could now write letters, keeping the lines straight, though he could not read what he wrote. His friends had been astonished at his facility in crossing the road near Victoria Station. His face had changed from the blank expressionless aspect of the blind man to one alive and responsive.

Section of Laryngology

President—W. M. MOLLISON, M.Chir.

[*November* 2, 1934]

DISCUSSION ON THE LARYNGEAL PARALYSES

Archibald Durward: I propose to confine my remarks to a brief account of the accepted central connexions within the medulla of the nerves which are distributed to the muscles and the mucosa of the larynx. The nerves involved are the vagus and the bulbar portion of the accessory. The classification of these nerves may be unfortunate and confusing; the bulbar portion of the accessory must be regarded functionally as a part of the vagus complex, concerned in the innervation of certain striped muscles along the course of the vagus in the neck. The association of the bulbar and spinal parts of the accessory is brief; from the viewpoint both of peripheral distribution and of central nuclear connexions the two are independent.

Of the various branches of the vagus complex in the neck, only two concern us in the present discussion, namely, the superior laryngeal nerve and the inferior or recurrent laryngeal nerve and some points about the so-called anastomosis between them in the laryngeal region will bear discussion. The actual pattern of the anastomosing rami of the two nerves varies, but such a connexion is to be regarded as constant. It has been suggested that some superior laryngeal fibres might be distributed in the field of the recurrent nerve, and that the superior laryngeal nerve might supply or assist in the supply of the muscles other than the cricothyroid. Certain specific muscles have even been given a definite supply from the superior laryngeal e.g. the interarytenoid muscle. There is no good or sufficient evidence for this view. Experimental work on animals[1] lends no support to the view that the internal branch of the superior laryngeal nerve is motor to laryngeal muscle. If fibres can be traced into some muscles in man from the superior laryngeal nerve or from the anastomosis between it and the recurrent, this need not mean that any such muscle is receiving a motor supply from the nerve concerned; such fibres may be sensory to muscle and mucosa. The older view that the recurrent nerve supplies all the muscles except the cricothyroid is the more acceptable to-day. The recurrent nerve is predominantly motor, but contains sensory fibres from the lower larynx as well. The superior is predominantly sensory and supplies only one muscle of the larynx.

A section of the medulla shows the nucleus ambiguus as a long column of cells deeply placed and adjacent to the spinal tract of the trigeminal. From this cell column fibres pass into the ninth, tenth and the bulbar portion of the eleventh nerve. The caudal part of the nucleus ambiguus is the centre for laryngeal movements and from the cells here the fibres travel towards their distribution in the larynx, viâ the bulbar portion of the eleventh nerve. The blood-supply of the nucleus ambiguus generally comes within the field supplied by the posterior inferior cerebellar artery. Sensory fibres from laryngeal mucosa travel in both laryngeal nerves to the tractus solitarius. Cell bodies are located in the vagal nuclei. The tractus solitarius also

[1] LEMERE, F., *Amer. Journ. Anat.*, 1932, li, 417.

receives afferents from the seventh and ninth nerves (taste). The nucleus of the tractus solitarius is placed immediately adjacent and its cells can deflect the impulse to the cord and brain stem nuclei for reflex purposes or towards the thalamus probably viâ the medial fillet.

Cell bodies may be found in and about the laryngeal muscles and along the course of the laryngeal nerves. Their nature is a matter of dispute; they may be aberrant receptor cell bodies or, again, peripherally placed parasympathetic neurones—most probably the latter.[1]

C. A. Joll: As early as 1885, when operations on the thyroid gland were still unusual, Jankowski [4] gave a description of laryngeal paralysis following total thyroidectomy, and Wölfler [8] in 1890 recorded six bilateral cases with four deaths. These publications induced Billroth [11] and Kocher [18] to systematize the operation of hemithyroidectomy, but this proved advantageous only because it resulted in the substitution of unilateral for bilateral paralysis. Socin and Reverdin avoided some of the dangers by resecting only the more prominent masses of thyroid tissue. Mikulicz's [17] melon-slice resection of the thyroid, though more successful in avoiding damage to the recurrent laryngeal nerves, proved even less satisfactory as a means of extirpating diseased thyroid tissue; Kocher's [16] method of resection-enucleation for thyroid adenomata fulfils most of the desiderata for avoiding nerve damage, though it is not applicable to all types of goitre.

Anatomy.—Either or both of the laryngeal nerves, superior and inferior (recurrent), may be damaged through thyroid operations. The superior laryngeal nerve often lies close to the inner side of the trunk of the superior thyroid artery, particularly at a point a short distance above the superior pole of the gland. In ligating the main artery, remote from the pole, the nerve may be inadvertently included. I have often identified it during preliminary operations for ligation of the superior thyroid artery—if touched, it gives a sharp painful sensation—and this itself may provide sufficient warning to enable one to avoid it. The best course, however, is to ligate the artery as close as possible to the pole of the gland.

The recurrent laryngeal nerve is comparatively seldom exposed in routine operations of the thyroid gland, because in most cases both lobes of the gland can be dislodged without disturbing the loose cellular tissue in which the nerve lies. I estimate that I expose the nerve, sufficiently to recognize it, in not more than 10% of all goitre operations and then usually at the stage when the inferior thyroid artery is to be ligated in continuity. It is only when the artery happens to be a little difficult to identify, or in a slightly unusual position, that the dissection necessary to expose the artery reveals the nerve also. It is of little practical value to know that in 65% the nerve lies behind the artery and in 26% in front of it, that in 9% it passes between its branches, or that it is inclined to be a little deeper on the left than right; it is sufficient to be aware that it may lie in any of these positions and to be alert particularly when approaching *the middle third of the lateral border of the gland.* By pulling the lateral lobes forward, no essential change occurs in the relationship of the nerve to the inferior artery or the thyroid gland, and therefore this manœuvre, if properly carried out, is perfectly safe, even though Crile [7] and others advise against it. The recurrent nerve lies at first outside the surgical or false capsule of the gland and is a little further from the trachea on the right than on the left; as it passes upwards it approaches the gland and penetrates the posterior layer only of the false capsule; the thin anterior layer of the capsule which is apposed to the postero-internal aspect of the gland is not perforated. Thus it follows that the recurrent nerve lies between the two layers into which the surgical capsule is divided posteriorly, while the inferior artery or its branches must perforce perforate both

[1] LEMERE, F., *Anat. Record*, 1932, liv, 389.

layers of this capsule. In all but a few exceptionally lowly placed inferior arteries, the nerve is at least half to three-quarters of an inch medial to the trunk of the artery at the point where the latter emerges from behind the carotid sheath. There is a rare anomaly of the recurrent nerve worthy of note which I have seen once only; similar cases have been described by Pemberton and Beaver [19], Milianitch [28], and others: the vagus may give off the recurrent nerve relatively high in the neck so that instead of hooking upwards behind the right subclavian it merely crosses the common carotid to reach the posterior border of the thyroid gland rather more transversely than usual, being, for this reason, more vulnerable.

Physiology.—The opinion held most widely in this country is that the intrinsic laryngeal muscles are supplied by the recurrent laryngeal nerve and that the cricothyroid is supplied by the external branch of the superior laryngeal. On the other hand Dilworth [13], Berlin and Lahey [12], New [29], Nordland [26], and others, claim that the arytenoideus and possibly other laryngeal muscles have a double nerve supply, i.e. derived from both the superior and inferior laryngeal nerves. None of them go so far as Exner [14] who believed that all the intrinsic muscles have a double nerve supply, though Berlin and Lahey [12] suggest that the internal laryngeal is usually the exclusive motor nerve to the arytenoideus.

A number of recent writers, New and Childrey [24], also Smith, Lambert and Wallace [23], have cast doubt on Semon's law [5] mainly because in none of the cases reported by the former investigators, and in only one reported by the latter, did the vocal cord pass from the median to the cadaveric position with lapse of time. The real crux of the discrepancy appears to me to lie in the essential difference between a slowly progressive lesion of the nerve such as is produced by an infiltrating neoplasm and the sudden traumatism inflicted during an operation. The latter must nearly always fail to provide the necessary conditions for a strict limitation of the damage to the abductor fibres. So far as purely laryngoscopic appearances are concerned my own experience accords closely with that of New and Childrey [24].

It is much more difficult to obtain a clear idea as to the incidence of pre- and post-operative laryngeal paralysis than would be expected. The reasons are:—

(1) Systematic pre-operative and post-operative laryngoscopic examinations have seldom been carried out in a series of cases.

(2) Such statistics as are available are based largely upon the patient's capacity to speak clearly. We know that complete unilateral paralysis, whether pre- or post-operative, is compatible with a normal speaking voice. On the other hand post-operative laryngitis and tracheitis are very likely to modify the voice without any gross paralytic changes.

Pre-operative paralysis.—In the case of malignant goitre, paralysis may be bilateral or unilateral, complete or incomplete, and is probably due to direct invasion of the nerve. In simple goitre paralysis is due to stretching of the nerve over a tense, bulky or rapidly growing nodule or cyst, but it may also follow the attacks of low inflammation common in and around nodular goitres, or be the result of hæmorrhage into an adenoma. In many cases it is impossible to explain the exact mechanism of the paralysis (cf. Layton's case [9] of bilateral paralysis in Graves' disease).

Very few reliable statistics are available. In 1,000 non-malignant goitre cases at the Mayo Clinic Matthews [1] found 262 in which there existed partial or complete, unilateral or bilateral paralyses, but he fails to differentiate between functional and organic lesions and it is clear that his criteria must be at fault to produce so high a figure. Feuz [20] in a series of 250 cases from Roux's Clinic found only one instance of pre-operative paralysis. In 1,000 cases from de Quervain's Clinic the pre-operative incidence was 3·1% (partial and complete). In a series of 422 cases

of my own examined by my laryngological colleagues at the Royal Free Hospital there was a total of 5 functional and 11 organic lesions (*see* Table I).

TABLE I.—LARYNGEAL EXAMINATION OF A SERIES OF CASES OF THYROID DISEASE AT THE ROYAL FREE HOSPITAL FROM JANUARY 1, 1930, TO SEPTEMBER 30, 1934

Type of disease	Number of cases examined by laryngoscope	Normal	Abnormal	Pre-operative	Post-operative
I. Primary thyrotoxicosis	200	195	5	4	1
II. Secondary thyrotoxicosis	98	92	6	4	2
III. Generalized nodular goitre ...	48	44	4	4	0
IV. Localized nodular goitre	63	60	3	3	0
V. Malignant goitre	4	3	1	1	0
VI. Colloid goitre	7	7	0	0	0
VII. Lymphadenoid goitre	2	2	0	0	0
Total ...	422	403	19	16	3

Statistics of post-operative laryngeal paralyses are largely invalidated by the absence of a systematic pre-operative and post-operative laryngoscopic examination. Kocher [6] had 9% in 900 cases, von Eiselsberg [15] 20% in 330 cases and de Quervain [2] 2·9% in 1,000 cases; most of these figures being subject to the proviso I have already made.

Feuz [20] reports a series of 250 cases from Lausanne examined strictly, before and after operation, with a 7·6% incidence of organic paralyses due to the operation.

In my series of 422 Royal Free Hospital cases the post-operative laryngoscopic findings were as follows. (It must be noted that, unless for special indications, most of the post-operative laryngoscopic examinations were made several days after the operation so that transient paralyses may have been missed.)

TABLE II.—PERCENTAGE INCIDENCE OF LARYNGEAL PARALYSIS

Total: ... ... 4·52%
(*a*) Pre-operative ... ... 3·79%
(*b*) Post-operative ... ... 0·73%

	Total incidence	Pre-operative	Post-operative
I. Primary thyrotoxicosis	2·5%	2%	0·5%
II. Secondary thyrotoxicosis	6·12%	4·08%	2·04%
III. Generalized nodular goitre ...	8·33%	8·33%	0·0%
IV. Localized nodular goitre ...	4·79%	4·79%	0·0%
V. Malignant goitre	25%	25%	0·0%

TABLE III.—ANALYSIS OF CASES OF LARYNGEAL PARALYSIS IN DISEASES OF THE THYROID GLAND

	Primary thyrotoxicosis 5 cases	Secondary thyrotoxicosis 6 cases	Generalized nodular goitre 4 cases	Localized nodular goitre 3 cases	Malignant 1 case
Functional paralysis	3 pre-operative	0	2 pre-operative		
Unilateral recurrent laryngeal nerve paralysis					
(*a*) Incomplete	0			1 pre-operative	
(*b*) Complete	1 post-operative	4 2 post- and 2 pre-operative	2 pre-operative	1 pre-operative	
Bilateral recurrent laryngeal nerve paralysis					
(*a*) Incomplete					1 pre-operative
(*b*) Complete		1 pre-operative		1 pre-operative	
Ext. laryngeal paralysis	1 pre-operative	1 pre-operative			

Prognosis.—Judd [3] states, rather vaguely, that the prognosis in immediate paralysis is good, but he appears to refer to the vocal recovery only. Feuz [20] reports that approximately 50% of the cases of complete and incomplete recurrent laryngeal paralysis which occurred in the Lausanne series (250 cases) disappeared within six to twelve months. The prognosis for pre-operative paralysis seems to be less favourable than for those which are the direct result of the operation.

In most cases the cord lies in the median position and remains there; only rarely does it pass into the cadaveric position. Apparently changes from the cadaveric to the medium position are not uncommon. Some paralyses are said to be transitory, but in the absence of repeated examination of the larynx this class of case is not easily definable and the symptoms ascribed to them may be due to local mechanical or inflammatory changes in the larynx rather than to actual nerve involvement.

Compensatory changes are fortunately the rule, so that functional recovery is commoner than restoration of nerve function—the contralateral cord swinging over the mid-line to lie close to its paralysed fellow.

In my series in one pre-operative case of incomplete bilateral paralysis the larynx is now quite normal. In three others, all of them pre-operative complete unilateral lesions, there has been no change except a trivial alteration from the cadaveric towards the median position. In two partial unilateral lesions there has been restoration to normal; in a third case there has been no change. The post-operative paralyses have persisted, though phonation is good in all three patients.

If prophylactic hæmostasis is made the primary principle of the operative technique, the nerve is, *ipso facto*, protected. There will then be no necessity for forcible plugging of the wound with gauze, or for the blind and hurried application of hæmostatic forceps in the depth of the wound. If by good fortune it is recognized during the operation that one recurrent nerve has been divided or included in a ligature, it is essential to remove the latter or to carry out primary suture of the divided nerve-ends, using for this purpose the finest silk on round-bodied needles, similar to those used in vascular suture. All attempts to practise *secondary nerve suture* for recurrent nerve lesions appear to me to have little chance of success. It is unlikely that the nerve-ends will be recognizable, or that they can be mobilized sufficiently to permit repair. Lahey [10] claims a success by this method, but his description of the result ten months later hardly bears out his claim. The same applies to the case of Horsley and Miller [25]. Colledge and Ballance [27] describe a successful bilateral anastomosis between the phrenic nerve and the divided inferior laryngeal in a woman of 54. Provided the distal end of the latter can be exposed and recognized for a certainty, this appears to me a more promising method than secondary nerve suture.

It is almost impossible at first to distinguish between bilateral laryngeal paralysis and tracheal collapse, but I am convinced that when the former is asserted to have cleared up quickly, following a tracheotomy, the actual lesion present was, in fact, confined to the trachea. I have had a case of this kind: Sudden stridor and cyanosis were noticed during an operation for thyrotoxicosis in a middle-aged man, and bilateral paralysis was diagnosed. A low tracheotomy opening made, but within a couple of days the patient was able to dispense with the tube. His cords were found to be sluggish and inflamed, but free from paralysis. No gross evidence of tracheal collapse had been detected at the time of the operation. I have no personal experience of the use of a permanent tracheotomy tube for these bilateral cases. The only proved case of bilateral paralysis which has arisen in my own practice (not, however, in the statistical series which I have presented in this paper), was of delayed onset, and the dyspnœic attacks which drew my attention to the case did not cause much distress until upwards of a fortnight after the operation. Possibly this was an instance of paralysed cords passing from the cadaveric to the

median position, as Lahey says is the rule. Crile [7] recommends the permanent use of a special type of valved tracheotomy tube (Waugh's) for these cases, and there are other forms of cannulæ available which carry the stream of air back above the adducted cords, thus permitting of improved respiratory freedom.

Réthi [21] has described a method for detaching the adductors of the cords from their insertions after laryngofissure and claims that his successes (15 out of 16) have been confirmed by Kahler and by Albrecht.

Lemere [22] in bilateral cases advocates inducing paralysis of the cricothyroids by injecting the external laryngeal nerves with alcohol. He asserts that by abolishing the tensor effect of these muscles the cords move apart and that breathing is much improved, though at the expense of phonation.

REFERENCES.

1 MATTHEWS, J., "Observations on the vocal cords in one thousand cases of goitre," *Journ. Amer. Med. Assoc.*, 1913, lxi, 10-13.
2 DE QUERVAIN, "Le goitre," Geneva, 1923. Editions "Atar."
3 JUDD, E. S., "Laryngeal function in thyroid cases," *Ann. Surg.*, 1921, lxxiii, 321-327.
4 JANKOWSKI, F., "Lähmungen der Kehlkopfmuskeln nach Kropfoperationen," *Deutsch. Ztschr. f. Chir.*, 1885, xxii, 164-205.
5 SEMON, Sir F., "Clinical remarks . . . recurrent nerves," *Arch. Laryng.*, 1881, ii, 197-222.
6 KOCHER, T., "Ueber ein drittes Tausend Kropfextirpationen," *Arch. f. Klin. Chir.*, 1906, lxxix, 786-791.
7 CRILE, G. W., "The thyroid gland," Philadelphia, W. B. Saunders and Co., 55-64.
8 WÖLFLER, A., "Zur chirurgischen Anatomie und Pathologie des kropfes und Nebenkropfes," *Arch. f. Klin. Chir.*, 1890, xl, 346-428.
9 LAYTON, T. B., "Bilateral abduction paralysis in exophthalmic goitre," *Proc. Roy. Soc. Med.*, 1920-21, xiv (Sect. Laryng., 55).
10 LAHEY, F. H., "Suture of the recurrent laryngeal nerve for bilateral abductor paralysis," *Ann. Surg.*, 1928, lxxxvii, 481-484.
11 BILLROTH, quoted by CROTTI, A., "Thyroid and thymus," London, 1923 (2nd Ed.)
12 BERLIN, D. D., and LAHEY, F., "Dissections of the recurrent and superior laryngeal nerves: the relation of the recurrent to the inferior thyroid artery and the relation of the superior to abductor paralysis," *Surg., Gyn. and Obst.*, 1929, xlix, 102-104.
13 DILWORTH, T. F. M., "The nerves of the human larynx," *Journ. Anat.*, 1921-1922, lvi, 48-52.
14 EXNER, "Die Innervation des Kehlkopfes," *Sitzungsb. der Kais. Akad. der Wissensch.*, 1884, lxxxix, 63.
15 VON EISELSBERG, A., "Die krankheiten den Schilddrüse," Stüttgart, 1901, F. Enke.
16 KOCHER, T., "Die Therapie des Kropfes," *Deutsch. Klinik.*, 1904, viii, 1115-1184.
17 MIKULICZ, J., "Beitrag zur Operation des Kropfes," *Wein. med. Woch.*, 1886, xxxvi, 1-6, 40-44, 70-73, 97-101.
18 KOCHER, T., quoted by HALSTED, "The operative story of goitre," *Johns Hopkins Hosp. Reports*, 1920, xix, 72-258
19 PEMBERTON, J. DE J., and BEAVER, G. M., "Anatomy of right recurrent laryngeal nerve," *Surg., Gyn. and Obst.*, 1932, liv, 594-595.
20 FEUZ, J., "Goitres bénines et paralysies récurrentielles," *Rev. de Laryngol., Otol., Rhinol.*, 1934, lv, 32-78.
21 RÉTHI, A., "Le traitement chirurgical de la fixation médiane bilatérale des cordes vocales," *Rev. de Laryngol., Otol., Rhinol.*, 1934, lv, 801-827.
22 LEMERE, F., "Innervation of the larynx. An analysis of Semon's law," *Ann. Otol., Rhinol. and Laryngol.*, 1934, xliii, 525-540.
23 SMITH, A. B., LAMBERT, V. F., and WALLACE, H. L., "Paralysis of the recurrent laryngeal nerves: A survey of two hundred and thirty-five cases," *Edin. Med. Journ.*, 1933, xl, 344-354.
24 NEW, G. B., and CHILDREY, J. H., "Paralysis of the vocal cords: A study of two hundred and seventeen medical cases," *Arch. Otolaryngol.*, 1932, xvi, 143-159.
25 HORSLEY, J. S., and MILLER, C. M., "Suture of the recurrent laryngeal nerve," *Southern Surg. and Gyn. Assoc.*, 1909, xxii, 161.
26 NORDLAND, M., "The larynx as related to surgery of the thyroid based on an anatomical study," *Surg. Gyn. and Obst.*, 1930, li, 449-460.
27 COLLEDGE, L., and BALLANCE, C. A., "The surgical treatment of paralysis of the vocal cord and of paralysis of the diaphragm," *Brit. Med. Journ.*, 1927 (i), 553-559, 609-612.
28 MILIANITCH, N., "Note sur une anomalie d'origine des gros troncs nés de la crosse de l'aorte accompagnée d'une anomalie de trajet au nerf recurrent droit," *Bull. et mém. Soc. anat. de Paris*, 1924, xciv, 230-233.
29 NEW, G. B., "The larynx in diseases of the thyroid," *Ann. Clin. Med.*, 1923, i, 262-265.

George Riddoch: Discussion still centres round the question of the central connexions of the laryngeal nerves. The consensus of opinion now is that the motor fibres arise from cells in the lower part of the nucleus ambiguus. This nucleus, which innervates the palate from its upper part and the pharynx from its middle

part, belongs to the ventrolateral column of nuclei, of which the fifth and seventh also form a part. These groups of cells are concerned with the striated muscles which are derived from the branchial arches, and constitute the special visceral efferent column. The fibres which are destined for the larynx leave the medulla by the accessory nerve but, through the internal ramus, soon join the vagus by which they travel to their terminations. Sensory impulses from the larynx conveyed by the superior and recurrent laryngeal nerves and vagus trunk, pass through the ganglion nodosum, in which their cells lie, to the dorsal vagus nucleus in the bulb. It is probable that the chief medullary end-station for non-sensory afferents is the tractus solitarius and its nucleus (visceral afferent column). These impulses are concerned with visceral reflexes, and, by means of a descending pathway, take part in the reflex activities of respiration and coughing (Ransom).

Causes of Laryngeal Paralysis.

Supranuclear: In Parkinsonism the only prominent disorder of laryngeal function is a low-pitched, monotonous voice. This common clinical condition is easily recognized by the mask-like face, bent posture of the body, slow movement, cog-wheel rigidity. Like hemiplegia, it is not in itself a disease, but a syndrome the result of lesions of the motor basal ganglia due to several causes, such as degeneration, in paralysis agitans; inflammation, in encephalitis lethargica and—rarely—in syphilis; or vascular, in cerebral arteriosclerosis.

In double hemiplegia and pseudobulbar palsy, similar dysphonia occurs. These disorders are uncommon and are usually found in the middle or late periods of life. They are distinguished by weakness, spasticity and the reflex changes characteristic of pyramidal lesions and facile emotionalism.

Nuclear: (1) Acute bulbar palsy.

(*a*) *Vascular*: Hæmorrhage with survival is rare. The usual lesions are thrombosis of the basilar artery, or of the posterior inferior cerebellar artery, due either to arteriosclerosis or to syphilis.

The onset is sudden but may have been preceded by other symptoms and signs of cerebral arteriosclerosis or syphilis, such as headache, giddiness, mental impairment, or ocular palsies.

With thrombosis of the basilar artery, symptoms vary with the severity of the block and are usually asymmetrical. Dysphonia, if present, is associated with dysarthria, dysphagia and sometimes dyspnœa. There is weakness of the face and the limbs on one or both sides, with reflex changes and commonly unilateral or bilateral sensory loss, especially to pain, heat and cold on the face, body, and limbs. Cerebellar and vestibular disorders such as ataxia, nystagmus, and vertigo are usually present.

Thrombosis of the posterior inferior cerebellar artery, a branch of the vertebral, which supplies a wedge-shaped area on the lateral aspect of the medulla as well as the inferior part of the cerebellum and the dentate nucleus, is a relatively common vascular lesion in this situation, and its syndrome is easily recognized. There is sudden vertigo, commonly diplopia from skew deviation, and staggering, with or without vomiting. Sometimes there is severe pain in the face and head on the side of the lesion, which may persist long after the acute phase is over. The limbs, especially the upper limb, on the side of the lesion, are incoördinate and hypotonic, and there is nystagmus. On the same side, the palate, pharynx and larynx may be paralysed with some dysphagia and dysphonia. Involvement of the descending sensory root and nucleus of the fifth and of the spinothalamic tract results in loss of sensibility to pain, heat and cold, and sometimes to light touch, although in less degree, on the face on the side of the lesion and on the trunk and limbs on the opposite side.

(*b*) *Inflammatory.*—Acute anterior poliomyelitis may attack the pons and bulb, giving rise to nuclear palsies, weakness of the limbs, and cerebellar defects. The death-rate is high from respiratory paralysis. It occurs in young people, and the onset of the nervous symptoms is usually preceded and accompanied by fever, malaise, pains, and meningeal signs. Excess of cells, chiefly lymphocytes (although polymorphonuclears are found in the acute stage), slight reduction of chlorides and slight increase in protein are found in the cerebrospinal fluid.

Ascending atonic paralysis, first described by Landry, which begins in the lower limbs and spreads to the upper limbs and trunk, may reach the brain-stem and give rise, amongst other symptoms, to weakness of the vocal cords. It is a rare event in epidemics of acute anterior poliomyelitis, but is most commonly due to severe polyneuritis from various causes, such as alcohol, diphtheria and other intoxications. I have recently seen a case which followed acute streptococcal infection of the tonsils in a boy aged 10 years, and because of respiratory paralysis, the patient, who is steadily recovering, was saved only by being kept continuously in a Drinker respirator for five weeks.

Disseminated sclerosis sometimes declares itself in young adults, with symptoms pointing to lesions in the pons and occasionally in the bulb. The combination of vertigo, ataxia, unilateral deafness, and external rectus and facial palsy with pyramidal signs of sudden onset is not very rare and with it there may be such bulbar symptoms as dysphonia and dysphagia. As with all acute or subacute lesions, if the patient survives, and this is the rule in disseminated sclerosis at this stage, improvement soon begins.

Encephalitis lethargica in the acute stage occasionally attacks the bulb and gives rise to laryngeal palsies. But they are commonly associated with distinctive features of the disease from lesions in the mid-brain and hypothalamus, such as diplopia and nystagmus, disorders of sleep-rhythm and involuntary movements.

(*c*) *Traumatic.*—Severe injuries of the bulb are almost always fatal. But with injuries of less severity, weakness of the laryngeal muscles, presumably from lesions of the nucleus ambiguus, is sometimes found in association with other bulbar signs.

Chronic bulbar palsy.—Progressive bulbar palsy : This disease of unknown origin is rare under the age of fifty. It attacks the motor nuclei of the bulb (almost always in the first instance the hypoglossal) so that by the time that the larynx is affected, dysarthria, especially for consonants, for which free movement of the tongue is necessary, has become evident. Fibrillation of the tongue can be seen from the first, but wasting comes later. The orbicularis oris, which is supplied through the seventh nerve from the hypoglossal nucleus, is affected at the same time. Later the palate, pharynx, larynx, lower part of the face, and the respiratory muscles become progressively paralysed and there may be spastic weakness of the limbs from pyramidal lesions. Dysphonia is associated with attacks of choking, dysphagia and nasal regurgitation of fluids.

Syringobulbia : The tumour process is rarely primary in this region and usually extends from the cervical enlargement of the spinal cord. Bulbar signs, which include dysphonia, dysphagia and dysarthria from destruction of the nucleus ambiguus, often unilateral or asymmetrical, are preceded by the characteristic analgesia and thermo-anæsthesia in the face, head, neck, upper limbs and chest.

Bulbar compression.—Tumours of the cerebellum from compression, invasion, or both, may lead to laryngeal palsy, but the associated manifestations of hydrocephalus and cerebellar disorders make the diagnosis clear. Should bulbar symptoms appear early, they usually indicate an infiltrating, gliomatous tumour.

Extracerebellar tumours, especially when posteriorly placed, compress the bulb and cerebellum as well as the cranial nerves which arise from the pons and medulla. Aneurysms of the basilar or vertebral arteries, if they are large enough, do the same, but they are uncommon and their symptoms are variable and asymmetrical. With

aneurysms a murmur can sometimes be heard on auscultation of the back of the head.

Weakness of the vocal cords in tabes may be due to lesions of either the nucleus ambiguus or the vagus.

Peripheral.—The vagus, along with other bulbar nerves, may be damaged within the skull or in the jugular foramen, by inflammation, for example, secondary to otitis or infective lateral sinus thrombosis or from syphilis, or by tumour. The ninth, eleventh or twelfth cranial nerves, because of proximity, may be injured with the vagus and various syndromes have been described.

The superior laryngeal nerve, along with the fibres for the palate and pharynx, leave the vagus at the ganglion nodosum, so that lesions at or above this level are associated with sensory loss in the larynx.

The superior laryngeal branch is rarely damaged alone, but injury of the recurrent laryngeal is the most frequent cause of unilateral paralysis of the vocal cord. The commonest causes of recurrent laryngeal paralysis are: in the chest, in addition to aortic aneurysm, mediastinal neoplasm, and enlarged glands—in Hodgkin's disease; and in the neck, enlarged glands, and cancer of the œsophagus.

Paralysis of individual muscles in the larynx is always due to local conditions, such as inflammation or growths.

Muscular.—Laryngeal palsy is frequently a symptom of myasthenia gravis. It is a disease of early adult life, and affects females more commonly than males. Although nervous lesions, except occasionally retrograde changes, are absent, the muscles which are usually earliest and most severely affected are those supplied by the brain-stem. Ocular palsies, such as ptosis, diplopia, squint, and weakness of the orbicularis oculi, commonly come first but are followed by paresis of the face, masseters, tongue, palate, pharynx, and larynx, with corresponding symptoms. All the voluntary muscles of the body may become affected, and life may be threatened or ended from feebleness of the muscles of respiration. The weakness temporarily diminishes or disappears with rest, and in consequence all voluntary muscles, including those of the larynx, should be used as little as possible.

Hysterical.—Aphonia is a well-known hysterical manifestation.

Spasm of the glottis.—Adduction is most common, but transient spasm of the abductors is sometimes a consequence of sudden fright or severe pain. Laryngismus stridulus usually occurs in children but is less common now that rickets has become rarer.

Adductor spasm also occurs in the tabetic laryngeal crises, in hydrophobia, and in tetany, and is the cause of the "cry" which often ushers in an epileptic fit.

Sir Frederick Hobday said that veterinarians operated for laryngeal paralyses with a different motive from that of operators on the human subject and he had now operated on about 4,000 cases. Sir Felix Semon had once asked him what became of the voice, and he had replied that if the operation was a success, his patient, although it could gallop without distress, was dumb. Sir Felix had then said that the operation would be useless for the human patient, for a man might not mind his inability to run but he did wish to talk. However, when the number of operations had reached about 1,500, he had found that a number of his patients did attempt to "talk," for they produced a muffled neighing sound, which was one of the means of knowing whether a horse had undergone an operation of this kind.

A point of agricultural importance was that Government premium stallions travelling the country must now be certified not to have had an operation on the throat. He himself did not believe that roaring was universally hereditary. Roaring usually occurred after a cold, pneumonia, strangles, or influenza, and the causes were mainly invasion of the throat glands by the streptococcus of Schultz.

In one case only of those upon which he had operated was the right side alone paralysed. In a few both sides were affected, but in by far the majority the left side alone was affected.

With animals the operation was solely a matter of utility. Whether the patient was a hunter or a cart-horse it was better to have this operation performed than for the animal to have to wear a tube.

After the operation he usually asked that the horse should be given three months' rest. The minimum should be six weeks, the object being to assure that the adhesions had become firm. The operation was a plastic one and merely consisted in stripping the ventricle of Morgagni. By stripping the ventricle two raw surfaces were made, and the vocal cord was gradually pulled back by the adhesions. In from twelve to fourteen days the external wound was healed and the horse was then kept quiet on the pillar-rein for another week.

Günther had carried out a similar operation in Germany a hundred years ago, but he had so many deaths that he had advised his students not to do it. In those days, however, there were neither anæsthetics nor antiseptics.